

# **A CLINICAL STUDY IN GASTRIC OUTLET OBSTRUCTION**

*Dissertation Submitted for*

**MS Degree (Branch I) General Surgery**

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**The Tamilnadu Dr. M. G. R. Medical University**

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## **CERTIFICATE**

This is to certify that this dissertation titled “ **A CLINICAL STUDY  
IN GASTRIC OUTLET OBSTRUCTION** ”submitted by  
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M.G.R. Medical University, Chennai in partial fulfillment of the requirement  
for the award of MS degree Branch I General Surgery, is a bonafide research  
work carried out by him under our direct supervision and guidance from 2009  
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I, **DR.ASHWIN.K** solemnly declare that the dissertation titled “**A CLINICAL STUDY IN GASTRIC OUTLET OBSTRUCTION**” has been prepared by me. This is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai**, in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

**Place: Madurai**

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## INTRODUCTION

Gastric outlet obstruction is described by Sir James Walton as “The stomach you can hear, the stomach you can feel and the stomach you can see”. This study has been taken up to review the changes in gastric outlet obstruction in view of changing trends in the management because of new drugs and investigatory modalities. The lack of uniformity in criteria in accepting a case of gastric outlet obstruction lead to differences in incidences and clinical features in different centres, still, any one of the following can be used to diagnose gastric outlet obstruction<sup>7</sup>.

1. Projectile vomitus of undigested food consumed previous day.
2. Visible gastric peristalsis (VGP).
3. Gastric succussion splash 3-4 hrs after last meal.
4. Palpable hypertrophied stomach.
5. A gastric residue of more than 500 ml in an adult.
6. An aspirate of more than 400 ml on saline load test.
7. Delayed emptying of stomach on barium meal studies.
8. Demonstration at operation of a grossly narrowed gastric outlet.

Cicatrised DU was the most common cause of gastric outlet obstruction, but due to wider usage of H<sub>2</sub> blockers and PPI's, better health care facilities with new investigations in armamentarium, its incidence is on decline and is on

decline and is replaced by carcinoma stomach which is detected early investigatory interventions and in some countries as part of screening programme.

## **AIMS OF THE STUDY**

1. To study various diseases presenting as gastric outlet obstruction in Govt Rajaji Hospital, Madurai.
2. To know the relationship with variables like age, sex, occupation and habits.
3. To evaluate diagnostic methods and various modalities of treatment pertaining to recovery.



# HISTORY

## **General Historical Survey :**

The physician of ancient and medieval times was often aware of the complications of gastric and duodenal ulcer, although he was rarely cognizant of their origin. Celsus (1<sup>st</sup> Century A.D) was apparently vaguely aware of gastric ulceration and referred to a “non-acid” diet as therapy. Galen (A.D. 131-201) and Paul of Aegina (A.D. 625 – 690) discussed the symptoms of haematemesis and melaena<sup>18</sup>.

In 1586 Marcellus Donatus of Mantua describes gastric ulcer at autopsy. In 1688 Muralto describes duodenal ulcer during autopsy which was followed by Morgagni who identified both gastric and duodenal ulcer during an autopsy in 1737<sup>2</sup>.

It was not until Mathew Baillie (1799) gave his accurate anatomic descriptions, coupled with clinical features, that the disease became a definite historical entity. Jean Cruveilhier was the first to investigate the subject in great detail (1829 – 1835)<sup>19</sup>.

## **History of specific surgical procedure**

### **Pyloroplasty**

This procedure was initially devised by Heineke in 1886 for treatment of congenital hypertrophic pyloric stenosis. Two years later similar procedure was described by Mikulicz and hence called “Heineke Mikulicz” pyloroplasty. John Finney performed widened pyloroplasty by anastomosing distal stomach to first and second parts of duodenum in 1903. In 1963 Weinberg revised Heineke Mikulicz pyloroplasty using single layer of suture to avoid infolding of tissues, luminal obstruction and decreased motility<sup>20</sup>.

### **Pyloromyotomy**

Ramsted in 1912 performed first pyloromyotomy for congenital hypertrophic pyloric stenosis<sup>20</sup>.

### **Gastrojejunostomy**

In 1881, Wolfler performed the first gastroenterostomy in an unresectable carcinoma of pylorus. Courvoisier reported the first posterior gastrojejunostomy in 1883. As obstructive symptoms were common after Wolfler’s method, Braun in 1892 and later Jaboulay (1892) introduced the addition of a lateral enteroanastomosis between the proximal and distal jejunal limbs to overcome regurgitant vomiting. Cesar Roux in 1908 transected jejunum whose proximal

end anastomosed to anterior gastric wall in front of transverse colon and proximal end to the side of jejunum<sup>1,20</sup>.

### **Billroth I gastrectomy**

First successful gastrectomy was done by Billroth in January 1881. The original Billroth operation consisted of gastric resection with gastroduodenal anastomosis<sup>1</sup>.

### **Billroth II gastrectomy**

In 1885 Billroth performs a successful distal gastrectomy and gastrojejunostomy for gastric cancer. Von Eiselberg (1889) was the first to modify the procedure by closing partially the gastric opening beginning at the lesser curvature end of the incision and anastomosing the jejunum with the greater curvature. Polya popularized the procedure of anastomosing the entire resected end of the stomach with the side of the jejunum, using a retrocolic segment. In Hofmeister's modification (1905), the jejunal loop was retrocolic and the afferent loop of jejunum was used to cover the closed portion of the transected end of the stomach, even though only the lower part was used for anastomosis<sup>13</sup>.

### **Antral exclusion procedures**

Von Eiselberg transacted the stomach proximal to unresectable pyloroduodenal carcinoma in 1895 closing transacted ends and performing a

gastrojejunostomy with the proximal stomach. In 1925 Devine transacted the stomach in its distal third and distal end closed. Proximal cut end was anastomosed with jejunum<sup>20,21</sup>.

## **Vagotomy**

Exner performed a subdiaphragmatic vagotomy on two patients in 1911. Latarjet in 1922 reported on 24 cases of incomplete vagotomy. Complete vagotomy was reported by Dragstedt and Owens in two patients through a trans thoracic approach in 1943. In 1945, Dragstedt published a series of 39 patients treated with truncal vagotomy, with remarkable results. This operation reduced the night gastric secretion by 50% - 60% but also caused gastric hypotonicity necessitating a bypass procedure.<sup>15</sup>

## **Highly selective or Parietal cell Vagotomy**

Griffith and Harkins realized that loss or surgical injury to the pylorus caused abnormal gastric emptying. They conceived the idea of denervating the parietal cell mass of the stomach without injuring the nerves to the antrum and pylorus. Their 1957 study was performed on dogs. Holle and Hart performed the first parietal vagotomy in man in 1967, but their addition of a drainage procedure detracted from the advantage of a more conservative vagotomy. Parietal cell vagotomy with preservation of nerves to the antrum and pylorus was first performed by Johnston and Wilkinson (1970) and Amdrup and Jensen (1970).<sup>14</sup>

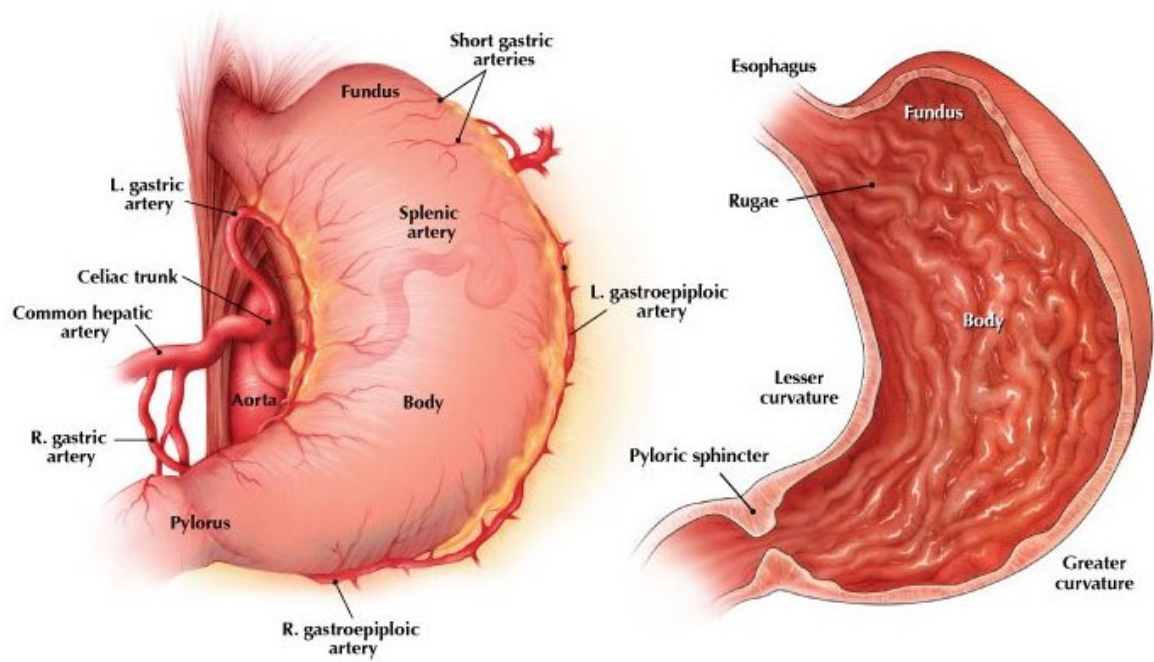
## EMBRYOLOGY AND ANATOMY

### **Development of the stomach and Duodenum<sup>22</sup> :**

The stomach, along with the first and second parts of the duodenum, are derived from the foregut. The stomach can be recognized as a fusiform dilatation at the end of the fourth week in the 4 mm embryo. At the 10 mm, stage the characteristic curvatures of the stomach are readily discernible. The stomach undergoes differential growth resulting in a considerable change in its shape and orientation. The original ventral border comes to face upwards and to the right and becomes the lesser curvature. The dorsal border now points downwards and to the left and becomes the greater curvature. The original left surface becomes anterior and the original right surface becomes posterior. The displacement and the rotation of the stomach has been variously attributed to its own growth changes, extension of the pancreatocenteric recess and pressure by the rapidly growing liver.

The part of the gut that gives rise to the duodenum forms a loop attached to the posterior abdominal wall by a mesentery (mesoduodenum). Later this loop falls to the right. The mesoduodenum fuses with the peritoneum of the posterior abdominal wall so that most of the duodenum becomes retroperitoneal. The lining epithelium of the duodenum proliferates and almost occludes the lumen by the 6<sup>th</sup> week, the channel is reestablished in the third month.

# STOMACH ANATOMY



Thus the stomach and proximal duodenum are supplied by branches of the Celiac artery and the distal duodenum by branches of the superior mesenteric artery.

## **ANATOMY**

Stomach is the most dilated part of the alimentary canal and is situated between the end of the oesophagus and the beginning of the small intestine. It lies in the epigastric, umbilical and the left hypochondriac regions of the abdomen. It has got variable shapes depending upon the volume and type of contents and position of the body. Its mean capacity at birth is 30 ml, 1 litre at puberty and about 1.5 litres in adult.

It is grossly divided into 5 parts<sup>5</sup>.

### **1. Cardia:-**

It is the area of stomach present immediately adjacent to cardiac orifice of stomach. It is the most fixed portion of stomach.

### **2. Fundus:-**

It is the upper convex dome situated above the level of the cardiac orifice. It is commonly distended with gas which is seen clearly in X-ray films under the left dome of diaphragm.

### **3. Body:-**

It is the portion of stomach lying between the fundus and a vertical line drawn from incisura angularis to greater curvature of stomach.

### **4. Pyloric antrum:-**

It extends from body to sulcus intermedius. The margin between body and antrum is not distinct externally but is clear from within by noting incisura angularis.

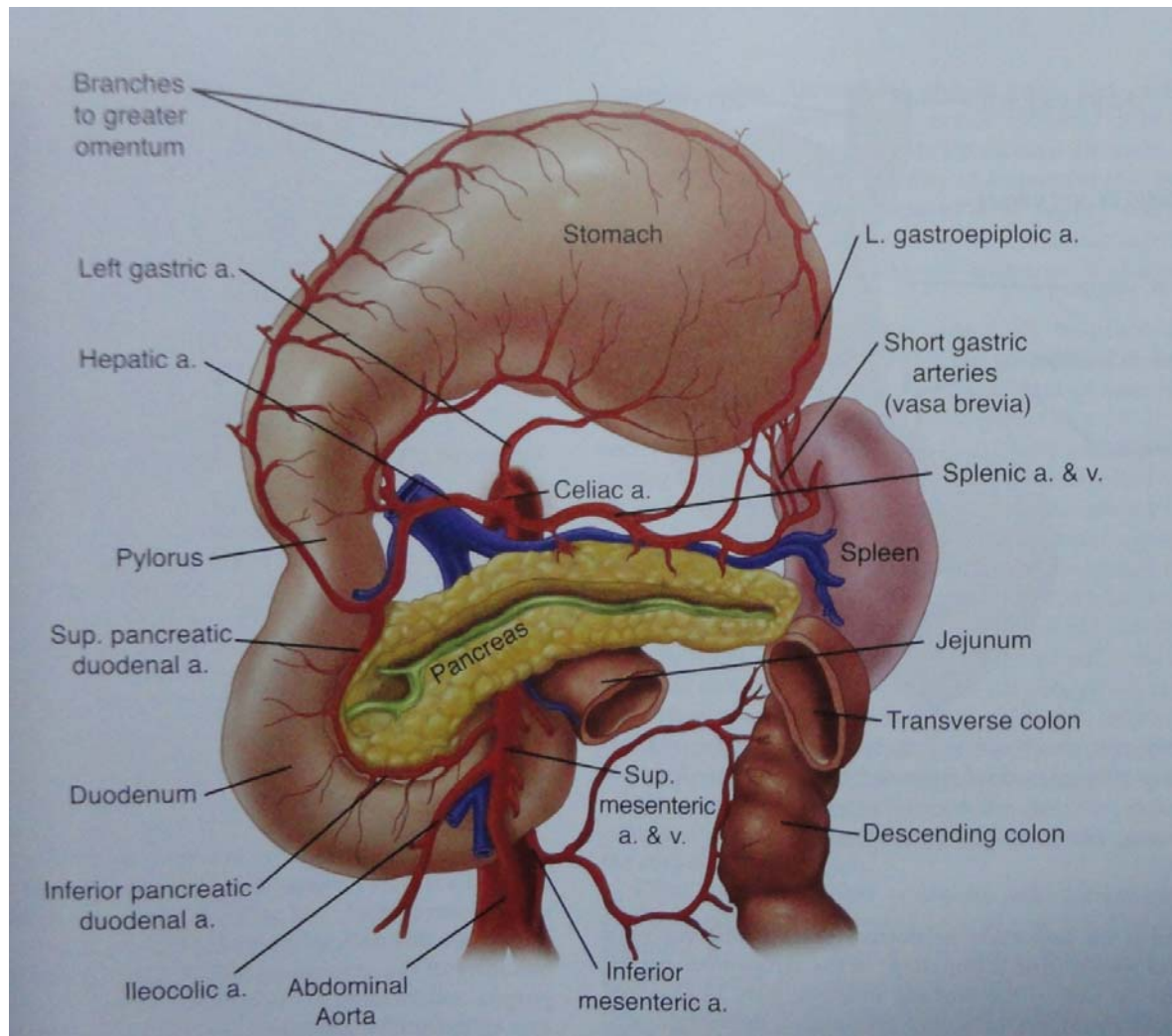
### **5. Pyloric canal:-**

Pyloric canal extends from the sulcus intermedius upto the pyloric opening which is identified on the surface by the pyloric vein of Mayo.

Stomach is covered by peritoneum all around except for small triangular area on posterior surface close to cardiac orifice and is called bare area of stomach. The stomach has 2 curvatures. The lesser curvature and the greater curvature. The lesser curvature is concave and forms the right border of the stomach. It provides attachment to the lesser omentum which contains neurovascular supply to stomach with common bile duct, portal vein and hepatic artery in the free margin. The most dependent part of the curvature presents the angular notch or incisura angularis. The greater curvature is convex and forms the left border of the stomach. It provides attachments to the greater omentum, gastrosplenic and gastrophrenic ligaments.



# BLOOD SUPPLY



Anteriorly, the stomach is related to the liver on the right side, and on the left, the diaphragm separates it from the base of the left lung, left pleura, pericardium, the 6<sup>th</sup> to 9<sup>th</sup> ribs and corresponding intercostals spaces. Posteriorly the lesser sac separates it from the diaphragm, left suprarenal gland, left kidney, splenic artery, pancreas, transverse mesocolon and splenic flexure of colon. The greater sac separates the stomach from the spleen.

### **Blood Supply of Stomach<sup>23</sup>:**

The stomach has a blood supply so extensive and inter connected that 3 of the 4 major nutrient arteries can be ligated without causing necrosis or significant dysfunction.

The stomach receives its blood supply through its two mesenteric borders by the left gastric (from the celiac axis), the right gastric and the right gastro – epiploic arteries (from the common hepatic artery) and the left gastro - epiploic and short gastric arteries (from the splenic artery).

The right and left gastric arteries run in the lesser omentum adjacent to the lesser curvature while the right and left gastro – epiploic arteries and vasa brevia run within the greater omentum adjacent to the greater curvature. These arteries supply the stomach by sending off specific anterior and posterior gastric branches that penetrate the stomach's muscular coat close to the lesser and greater curvature. On reaching the submucosa, these branches ramify

extensively throughout the entire submucosa. Maintaining a relatively larger caliber, these submucosal ramifications anastomose frequently with each other to form the sub-mucosal plexus which consists of both arteries and their venous counterparts. Independent branches from the submucosal plexus supply the mucosa everywhere except in the lesser curvature which receives branches directly from the right and left gastric arteries.

The gastric veins commence as straight vessels between the mucosal glands and these drain into the sub-mucosal veins. They then accompany their corresponding arteries to ultimately drain into the splenic and superior mesenteric veins.

### **Nerve supply<sup>8</sup>**

Parasympathetic supply is derived from Vagus. The left and right vagus nerves descend parallel with the esophagus and contribute to a rich external esophageal nerve plexus between the level of the tracheal bifurcation and the level of the diaphragm. From this plexus, two vagal trunks, anterior and posterior, form and pass through the esophageal hiatus of the diaphragm. Each trunk subsequently separates into two divisions. Anterior vagus gives off hepatic branch, which supplies liver, gall bladder and pyloric antrum. It gives off fundic branch and continues as nerve of Latarjet giving off branches to acid and pepsin secretory areas of stomach. At a point 5-7 cms proximal to pylorus it divides into branches and the appearance is described as 'crow's foot'. Posterior

vagus gives off a celiac branch to celiac ganglion and usually also a branch to supply the antrum. Criminal nerve of Grassi is a branch from posterior vagus. Vagus is motor to the gut (via Aurbach's plexus) and secretomotor to the glands (via Meissner's plexus).

The thoracic splanchnic nerves are formed mostly of preganglionic fibers from the intermediolateral cell column of the spinal cord at levels T5-T10. These terminate within the celiac ganglia, where preganglionic fibers synapse upon the collateral ganglion cells. Postganglionic fibers from the celiac ganglia reach the stomach and duodenum via their blood supply. Afferent fibers for the sense of pain from the organs supplied by the celiac artery (including the stomach) pass through the celiac plexus and thoracic splanchnic nerves to the sympathetic chains. They reach spinal nerves at thoracic levels T5-T10. The cell bodies of these fibers are found in dorsal root ganglia at those levels. The sympathetic nerve pathway is the connection of the stomach to the spinal cord via sympathetic neurons and dorsal root ganglia. Efferent fibers start in the celiac ganglia and end in the gastric wall as target cells. Afferent fibers whose peripheral processes originate in the gastric wall have cell bodies in the dorsal root ganglia, with central processes entering the spinal cord.

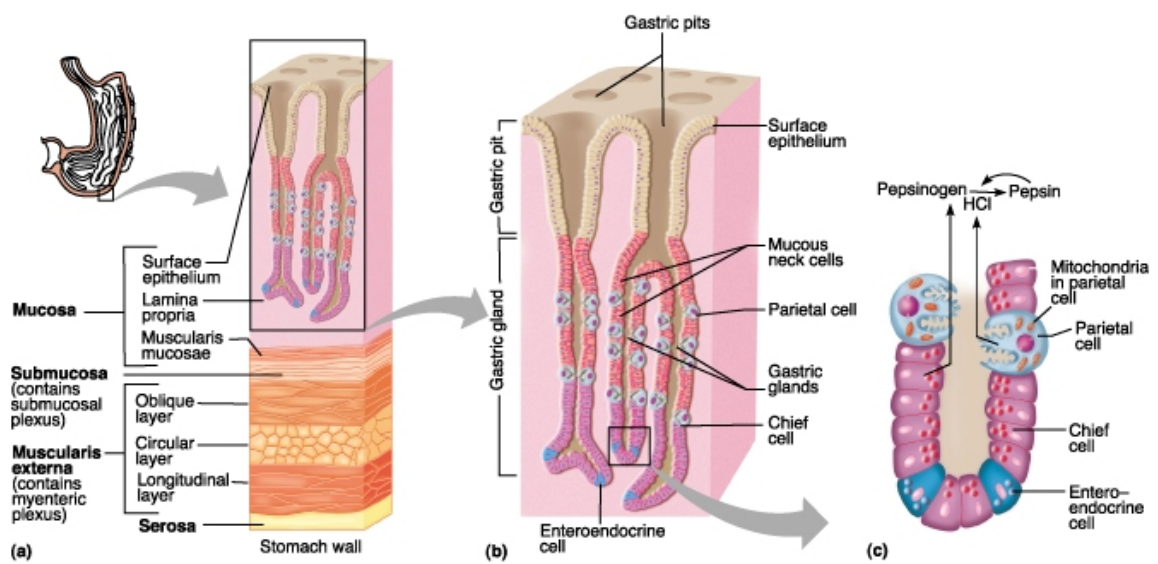
## **Lymphatic Drainage of stomach<sup>4, 23</sup>**

The lymph channels follow the artery. The lymph originates primarily in the mucosa and drains into the lymphatic sub-mucosal plexus which is as rich and extensive as that of the arterial and venous sub-mucosal plexus. They also anastomose with the sub-mucosal plexus of the oesophagus while the duodenum is relatively devoid of sub-mucosal plexus and therefore sub-mucosal spread of carcinoma into the duodenum is unusual. The sub-mucosal plexus then drains into the sub-serosal plexus just beneath the peritoneum. From this point the lymphatic drainage continues through extrinsic channels which are divided into 4 sets

- **Superior gastric** group drains lymph from the upper lesser curvature into the left gastric and paracardial nodes.
- **Suprapyloric group** of nodes drains the antral segment on the lesser curvature of the stomach into the right suprapancreatic nodes.
- **Pancreaticolienal group** of nodes drains lymph high on the greater curvature into the left gastroepiploic and splenic nodes.
- **Inferior gastric and Subpyloric group** of nodes drains lymph along the right gastroepiploic vascular pedicle.

All four zones of lymph nodes drain into the celiac group and into the thoracic duct. Although the aforementioned lymph nodes drain different areas of the stomach, it remains widely recognized that gastric cancers may

# STOMACH HISTOLOGY



metastasize to any of the four nodal groups regardless of the cancer location. In addition, the extensive submucosal plexus of lymphatics accounts for the fact that there is frequently microscopic evidence of malignant cells several centimeters from the resection margin of gross disease.

### **Histology<sup>24</sup>**

Stomach can be divided into three areas, cardia, fundus and pylorus according to histology. Gastric mucosa protruding as rugae into the lumen is covered with gastric glands and microvilli. Parietal cells the major component, producing acid and intrinsic factor. Between these are chief cells which produce pepsinogen. Endocrine or argentaffin cells are scattered throughout the stomach. Gastric glands occur throughout the stomach and contain parietal cells in fundus which are largely replaced by mucus cells in cardia and gastrin producing endocrine cells in pylorus, which can be seen only on immunofluorescence.

Stomach wall is composed of circular, oblique and longitudinal muscles. The pylorus sphincter is circular smooth muscle. Outer most layer is the serosa.

### **First part of the Duodenum <sup>23</sup>:**

The first part of the duodenum is about 5 cms long and the most movable of the 4 parts of the duodenum. It begins at the pylorus and ends at the neck of the gall bladder. It is covered with peritoneum over the whole of the anterior

portion. But is devoid of peritoneum posteriorly. It is related above and in front to the quadrate lobe of the liver and gallbladder ; above and posteriorly to the epiploic foramen; behind with the gastro duodenal artery, the bile duct and portal vein ; and below and behind with the head and neck of the pancreas. It is supplied by the hepatic, gastro-duodenal and pancreaticoduodenal arteries. The lymph vessels run anteriorly and posteriorly to end in the pyloric nodes, which are present on the anterior and posterior parts of the pancreaticoduodenal groove.

Microscopically it has long slender villi, lined by columnar epithelium. Brunner's gland is situated between muscularis mucosae and muscle layers and secretes mucin.



## PHYSIOLOGY

Gastric glands secrete about 250ml of gastric juice daily. HCl secreted by parietal cells kills many bacteria, aids protein digestion, provides necessary pH for pepsin to start protein digestion and stimulates the flow of pancreatic and bile juice. Gastric mucosa is protected from HCl by

1. Mucus secretion: Mucin secreted by neck and surface cells forms a flexible gel layer which exhibits a diffusion coefficient for  $H^+$  that is one fourth of that of  $H_2O$ . Acid and pepsin containing fluid exits the gastric glands as jets directly entering the lumen without contacting surface epithelial cells.
2. Bicarbonate secretion: Epithelial cells secrete  $HCO_3^-$  creating an essentially neutral micro environment.
3. Epithelial barrier: Tight junctions prevent backflow of HCl.
4. Mucosal blood flow: Rich blood supply removes back diffused acid and provides  $O_2$ ,  $HCO_3^-$ , and nutrients.

### Regulation of Gastric Secretion<sup>24,25</sup>

1. Cephalic phase: Accounts for one third to half of gastric secretion. Stimulation of anterior hypothalamus and parts of adjacent orbital frontal cortex increases vagal efferent activity by sight, smell or thought.

2. Gastric phase: due to stretch and chemical stimuli to receptors in the wall of stomach.
3. Intestinal phase: Presence of food in the duodenum causes the stomach to secrete small amounts of gastric juice secondary to gastrin secretion from the duodenum.

### **Gastric motility** <sup>25</sup>

Gastric pacemaker is present in circular fibres of fundus. Following a meal stomach exhibits receptive relaxation lasting for few seconds. Following this adaptive relaxation occurs which allows proximal stomach to act as reservoir. Antral systole occurs about every 20 seconds and lasts for about 10 seconds. Partial contraction ahead of advancing gastric contents prevents solid masses entering duodenum. Most of peristaltic activity is found in distal stomach (antral mill) and proximal stomach demonstrates only tonic activity. Antral contraction against a closed sphincter is important in the milling activity of stomach. Gastric emptying is delayed by protein and fat rich or highly osmotic diet, fear and vagotomy.

Intrinsic factor necessary for vitamin B12 absorption is secreted by parietal cells of gastric mucosa. Following total gastrectomy patients should be supplemented with parenteral cyanocobalamine to overcome Vitamin B12 deficiency.

# CAUSES AND MANAGMENT OF GASTRIC OUTLET OBSTRUCTION

## **Gastric Outlet Obstruction Secondary to Chronic Duodenal Ulcer :**

About 10 –15% of cases of chronic duodenal ulcer develop gastric outlet obstruction and is the most common cause of gastric outlet obstruction<sup>28,37</sup>.

### **Pathology<sup>26,27,33</sup>:**

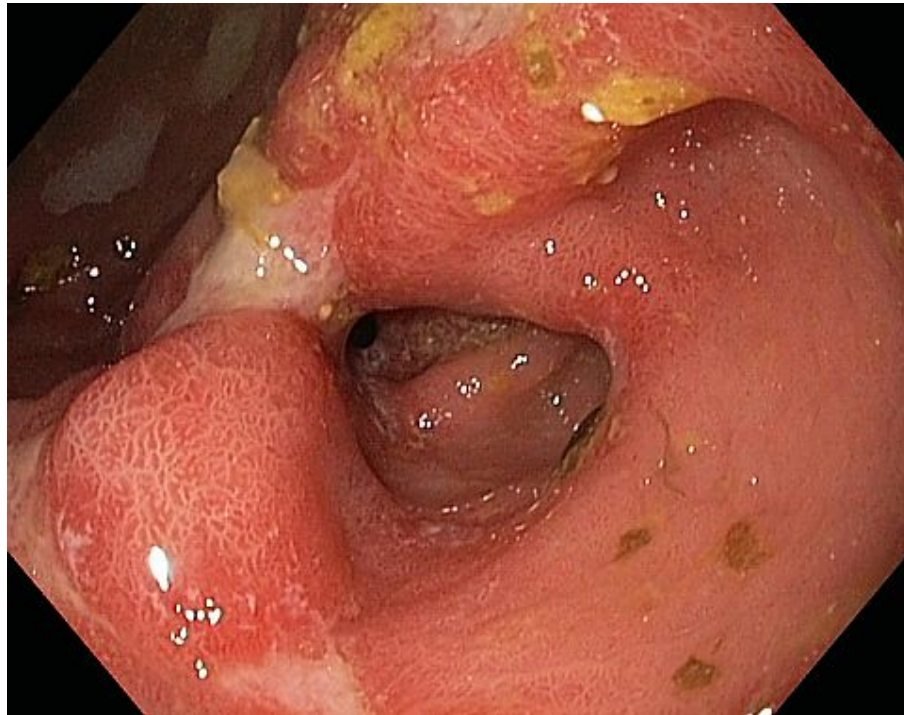
The pathogenesis of duodenal ulcer is not clear and no single theory explains all types of lesions. The various factors involved in ulcer formation include (a) hyper secretion of acid which is associated with 40% of the cases; (b) impaired mucosal defence caused by various agents like NSAID, smoking, stress and H-pylori, even though the acid secretion is within normal limits (c) genetic factor – there is evidence to show that the duodenal ulcer runs in families and 40% of the people with this autosomal dominant characteristic develop duodenal ulcer.

The obstruction is caused by chronic cicatrisation of the duodenal ulcer in which the scar contracture gradually narrows the lumen.

### **Clinical Features<sup>28,37</sup>:**

There is a long preceding history of pain abdomen with loss in periodicity. As stenosis progresses and as the stomach becomes both dilated and

## **PYLORIC STENOSIS DUE TO CHRONIC DUODENAL ULCER**



decompensated, the actual character of the pain is altered. It becomes a generalized upper abdominal discomfort that builds during the day as the stomach fills and is then relieved by a massive vomit, which is sometimes self induced. The vomiting is projectile and contains food particles eaten a day or two previously. When vomiting becomes copious and frequent, the patient may complain of anorexia, coated tongue, thirst and weakness. In extreme cases, the patient may present with tetani, convulsions, mental disturbances or coma. Weight loss is frequently present due to reduced intake.

On examination, patient may be dehydrated. A gastric splash can be heard 3-4 hours after the last meal or drink. Visible gastric peristalsis can be observed passing from left to right . Rarely duodenal ulcer can produce an inflammatory mass with the head of the pancreas, adjacent omentum or the hepatic flexure of the colon.

### **Investigations<sup>35,36</sup> :**

Barium meal examination and endoscopy are done to confirm the diagnosis.

### **Pre-operative Management<sup>28,30</sup> :**

The patient is adequately hydrated and the electrolyte imbalance is corrected. Gastric lavage with normal saline is given twice a day. Patient is put on liquid diet and H<sub>2</sub> receptor blockers. Within a week, the inflammatory edema

subsides and the gastric muscle tone improves. Ascorbic acid is given parenterally as patients are on dietary restrictions for a long period. Anemia, if present, is corrected with whole blood transfusion.

### **Endoscopic Intervention<sup>34</sup>**

About 85% of gastric outlet obstruction cases are amenable to dilatation and 80% of these had immediate relief of symptoms. Only 40% of these sustained improvement after three months. The normal pyloric canal is 15-20 mm in diameter and dilates to 25 mm without difficulty. If a standard endoscope (11-12 mm) couldn't be passed, pyloric stenosis is probable. A pyloric ring of less than 6 mm is generally associated with symptoms of gastric outlet obstruction.

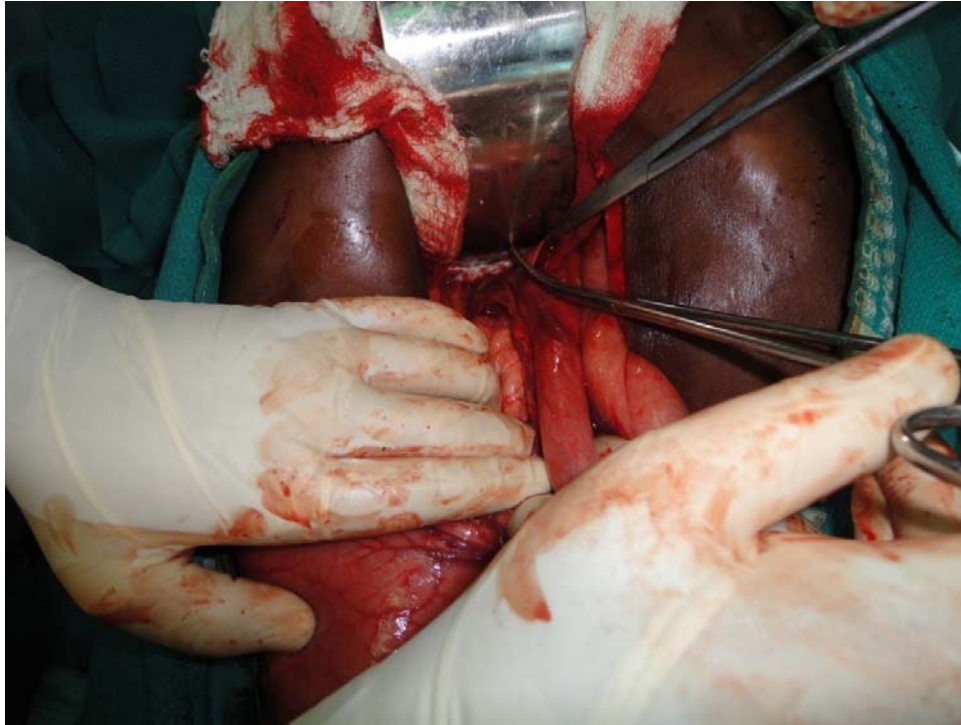
#### **a. Through the scope balloon dilatation**

A well lubricated balloon is passed through the biopsy channel. Balloon inflated to maximum pressure in stenosed area with water or diluted contrast medium using a pressure gauge. Pressure is maintained for 1 minute and repeated for 3-4 times. Duodenal bulb beyond is inspected.

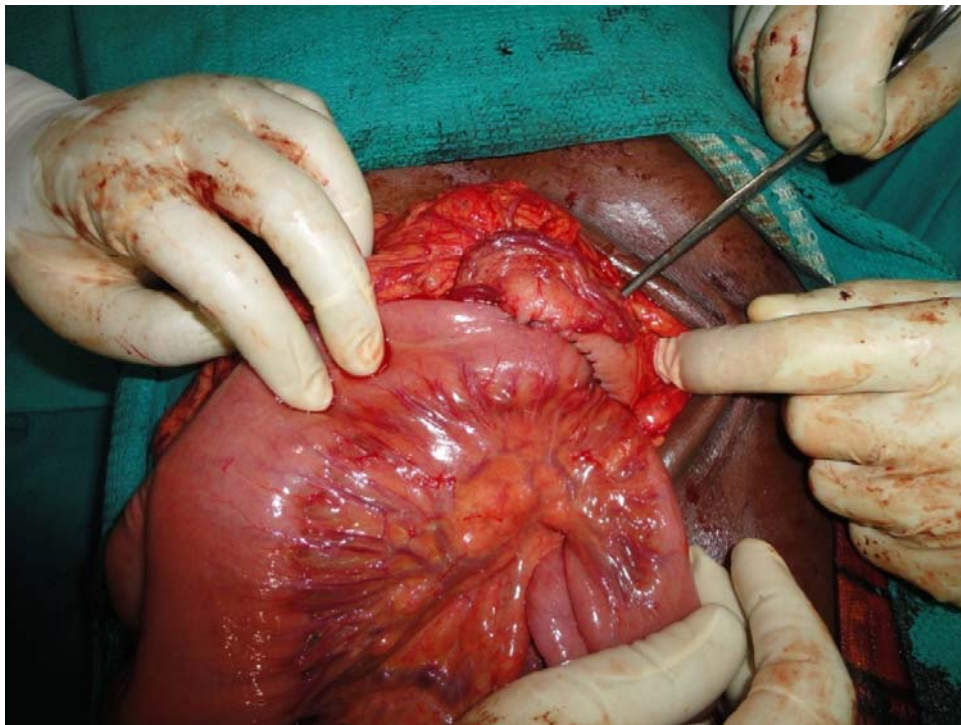
#### **b. Over the wire balloon dilatation**

A guide wire is advanced far enough in strictured area. Dilatation is done under fluoroscopic guidance using dilute radiographic contrast medium. Pyloric ring and duodenal bulb are examined endoscopically.

Post procedure contrast study is done to rule out perforation.



**Anterior Vagus dissected**



**Posterior Gastrojejunostomy**

**Surgery :**

Procedures devised for treatment of duodenal ulcers have common aim of excluding damage effects of acid from the duodenum. This has been achieved by diversion of the acid away from the duodenum, reducing secretory potential of stomach or both <sup>1</sup>.

The procedure of choice is between vagotomy and antrectomy and vagotomy with drainage procedure. If disease in duodenum is severe and stump cannot be closed, vagotomy with drainage procedure is treatment of choice. If disease in duodenum is not severe and duodenal stump can be closed, antrectomy with Billroth II anastomosis with vagotomy is treatment of choice. Truncal vagotomy with gastrojejunostomy preserves gastric reservoir and can be done with a lower risk.

**Truncal vagotomy <sup>1</sup>:**

Introduced in 1943 by Dragsted and for many years, combined with drainage was main stay of treatment of duodenal ulcers. Principle of this procedure being denervation of stomach from vagus reduces maximal acid output by approximately 50%. As vagus is motor to antropyloroduodenal segment, denervation results in gastric stasis in substantial number of patients and hence should be accompanied by a drainage procedure. It has 0.5-0.9%



mortality rate and 2-7% ulcer recurrence rate. There is delayed emptying of solids and early emptying of liquids.

### **Highly selective vagotomy <sup>1</sup>:**

Devised by Johnston and Amdrup in 1968 in which only parietal cell mass of stomach was denervated. This proved to be the most satisfactory operation for duodenal ulceration, with a low incidence of side effects and acceptable recurrence rates. Operative mortality was lower than any devised procedure in all probability because gastrointestinal tract was not opened. Mortality rate was < 0.2% and rate of ulcer recurrence was 2-10%.

### **Truncal vagotomy and antrectomy<sup>1</sup>:**

In addition to truncal vagotomy, antrum of stomach is removed thus eliminating the source of gastrin. Gastric remnant was anastomosed to the duodenum. Recurrence rate was extremely low (1%) however operative mortality was higher than other procedures (1%).

### **Drainage procedures <sup>3</sup>:**

- Pyloric dilatation: A balloon 15mm in length, may be positioned endoscopically and inflated to 45psi for 10 minutes. Alternatively a finger introduced across the pylorus through a small gastrostomy opening for dilatation of pylorus can be done. Gastrostomy is then closed.

- Pyloromyotomy: An incision made over anterior surface of stomach from 1-2 cms proximal to 1 cm distal to pyloric ring allowing the mucosa to protrude through the incision.
- Pyloroplasty: Most commonly performed is Heineke-Mikulicz procedure. Incision is made on anterior surface in a longitudinal direction from 2 cm distal to pyloric muscle to 3 cm proximal to pylorus. Closure of pylorus is done vertically in order to minimise narrowing of lumen.
- Finneys pyloroplasty: Used when scarring has involved pylorus and duodenal bulb and would not permit tension free patulous Heineke Mikulicz pyloroplasty. Finneys pyloroplasty is a side to side gastroduodenostomy performed after mobilising duodenum. Inverted U shaped incision made and gastroduodenostomy is done.
- Gastroenterostomy: Most dependent part of stomach is anastomosed to jejunum through isoperistaltic, retrocolic, shortloop, oblique, posterior gastrojejunostomy.

### **Carcinoma of the Stomach<sup>1,17,32</sup>:**

Gastric carcinoma when present in the pyloric region produces gastric outlet obstruction. 64% of carcinoma stomach occurs in the pyloric region, while 27% occurs in the body, 6% in the fundus and cardia and 3% involves the whole stomach.

## **Risk Factors :**

### 1. Nutritional

- a. Diets high in salt and smoked food
- b. High nitrate consumption
- c. Low dietary Vit A and C
- d. Poor drinking water

### 2. Genetic factors

- a. Male gender
- b. Black race
- c. Type A blood group
- d. Family history

### 3. Occupational

- a. Metal workers and miners
- b. Workers exposed to wood or asbestos dust
- c. Rubber workers

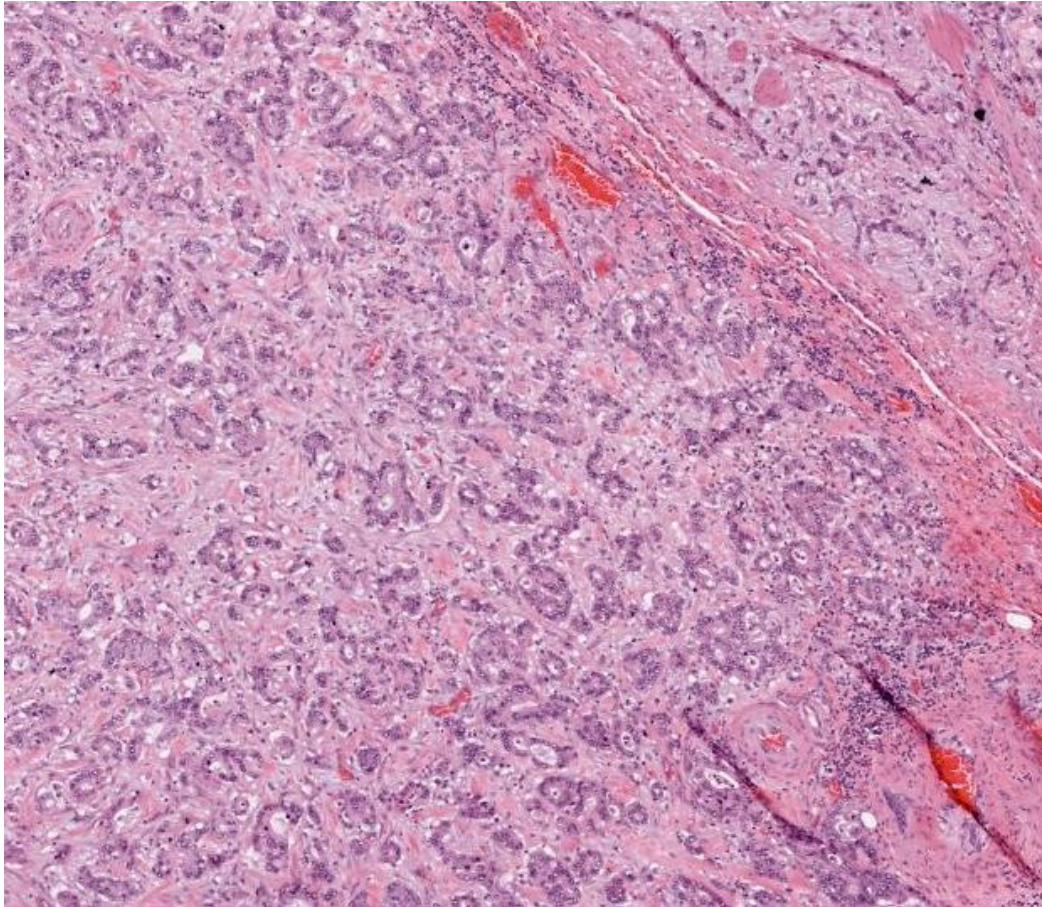
### 4. Cigarette smoking

### 5. H pylori infection

### 6. Precursor lesions

- a. Villous adenoma
- b. Intestinal metaplasia
- c. Menetrier's disease

## **INTESINAL TYPE OF GASTRIC ADENOCARCINOMA**



7. Prior radiation therapy
8. Epstein Barr virus infection

Pathological classification:

**1. Lauren's classification**

- a. **Intestinal form:** Occur more commonly in older patients, is found in geographic areas where there is high incidence of gastric cancer. They have tendency to form glands and tend to spread hematologically to distant organs.
- b. **Diffuse form:** More common in younger patients, tend to spread transmurally and by lymphatic invasion.

**2. Borrmann's classification:**

- Type I:** Polypoid or fungating
- Type II:** Ulcerated lesion with elevated borders
- Type III:** Ulcerated lesion with gastric wall infiltration
- Type IV:** Diffusely infiltrating
- Type V:** Unclassifiable

**3. Broder's classification:**

- a. Well differentiated
- b. Moderately differentiated
- c. Poorly differentiated
- d. Anaplastic

#### 4. **WHO classification** of adenocarcinoma

- a. Tubular
- b. Mucinous
- c. Papillary
- d. Signet ring cell type

#### Modes of spread:

- Direct infiltration: to adjacent structures by continuity and contiguity
- Lymphatic spread: spreads to surrounding lymphnodes in adjacent areas of stomach
- Transcelomic spread: deposition of tumor cells within the peritoneal cavity once serosa is breached. This can present as Krukenberg's tumor or as Blumer shelf.
- Haematogenous spread: mainly to liver, lungs, peritoneum, omentum, pancreas, adrenal glands and to the skin.

#### Clinical features:

Men are commonly affected more than women in a ratio of 2:1. The highest incidence of the disease occurs between the ages of 55 and 65 years. Vague epigastric discomforts for a period of 6 to 12 months, rapid weight loss, anorexia, abdominal pain are the main presenting features. Approximately 10% of patients present with one or more signs of metastatic disease. Common

pointers of distant metastasis are palpable supraclavicular lymphnode (Virchow node), mass palpable in rectal examination (Blumer shelf), a palpable periumbilical mass (Sister Mary Joseph node), enlarged left axillary lymphnode (Irish node), ascites, jaundice or a liver mass.

The absence of physical signs in a patient with symptoms does not exclude a malignant tumor of the stomach and the mere presence of a palpable mass does not indicate inoperability.

### **T N M Staging of Gastric Carcinoma :**

#### **Primary tumor (T)**

T<sub>1</sub> Tumor invades lamina propria or submucosa

T<sub>2</sub> Tumor invades muscularis propria or subserosa.

T<sub>2a</sub> Tumor invades muscularis propria.

T<sub>2b</sub> Tumor invades subserosa

T<sub>3</sub> Tumor penetrates serosa (visceral peritoneum) without invasion of adjacent structures.

T<sub>4</sub> Tumor penetrates the serosa and invades adjacent structures.

### Regional lymph nodes (N)

N<sub>0</sub> No Metastasis to regional lymph nodes.

N<sub>1</sub> Metastasis in 1-6 lymph nodes.

N<sub>2</sub> Metastasis in 7-15 lymph nodes.

N<sub>3</sub> Metastasis in >15 lymph nodes.

### Distant Metastasis (M)

M<sub>0</sub> No distant metastasis

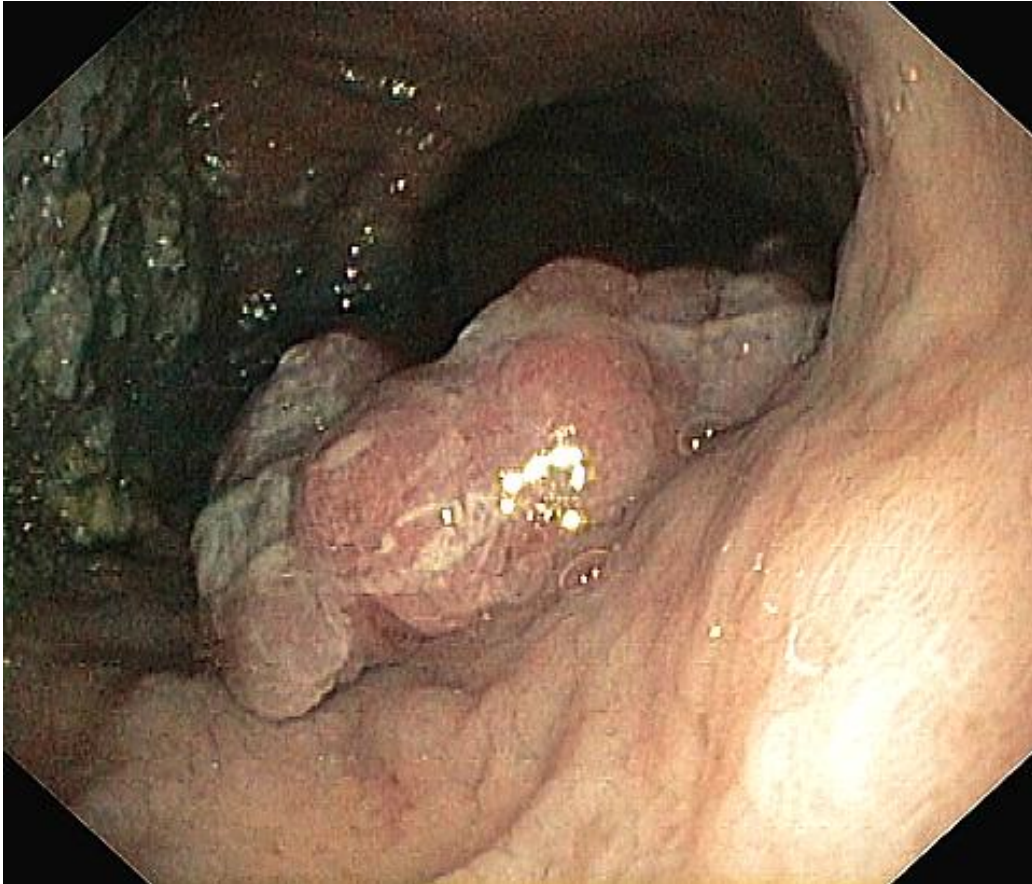
M<sub>1</sub> Distant metastasis

### Stage grouping

Stage 0	Tis	N0	M0
Stage IA	T1	N0	M0
Stage IB	T1	N1	M0
	T2a/b	N0	M0
Stage II	T1	N2	M0
	T2a/b	N1	M0
	T3	N0	M0
Stage IIIA	T2a/b	N2	M0
	T3	N1	M0
	T4	N0	M0
Stage IIIB	T3	N2	M0
Stage IV	T4	N1-3	M0
	T1-3	N3	M0
	Any T	Any N	M1



## **OGD SHOWING GROWTH IN A CASE OF CARCINOMA STOMACH**



## **5 year survival rates after gastrectomy with complete resection and >15**

### **lymph nodes examined**<sup>17</sup>

Stage IA     78%

Stage IB     58%

Stage II      34%

Stage IIIA   20%

Stage IIIB   8%

Stage IV     7%

### **Investigations:**

- Complete blood cell and platelet counts, blood urea, serum creatine, serum electrolytes.
- Chest radiography
- Computed tomography of abdomen and pelvis for overall staging of disease. Major limitation as a staging tool are in the evaluation of Early gastric cancers and small (<5 mm) metastasis on peritoneal surface of liver.
- Esophagogastroduodenoscopy to confirm diagnosis and for biopsy from the tumor.

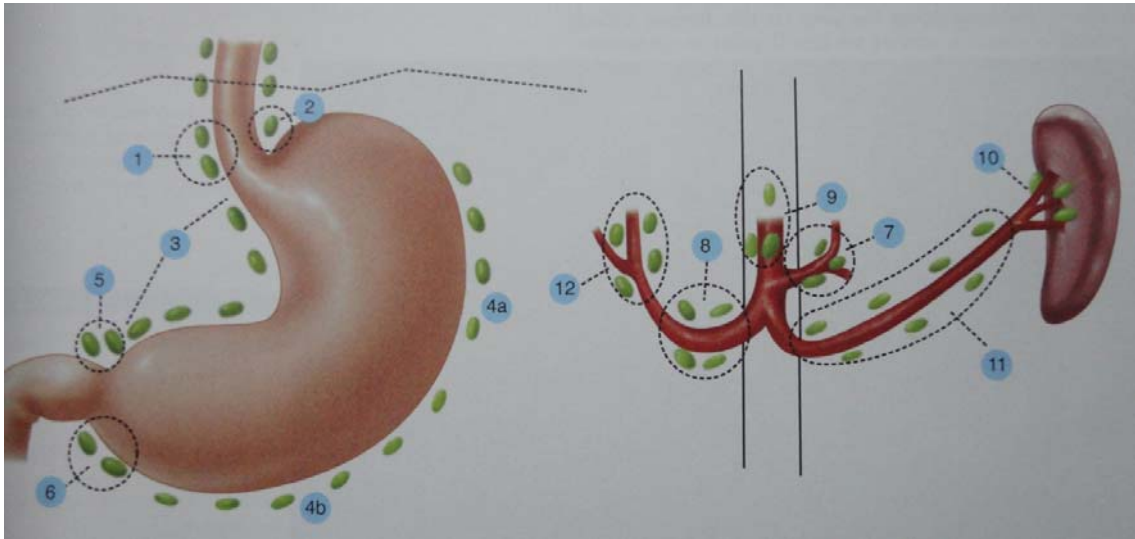
- Barium meal study: Irregular persistent filling defect with with short history suggests carcinoma.
- Staging laparoscopy.
- Positron emission tomography to detect CT occult metastasis and may be used to assess response to neoadjuvant therapy.

### **Treatment of carcinoma stomach**

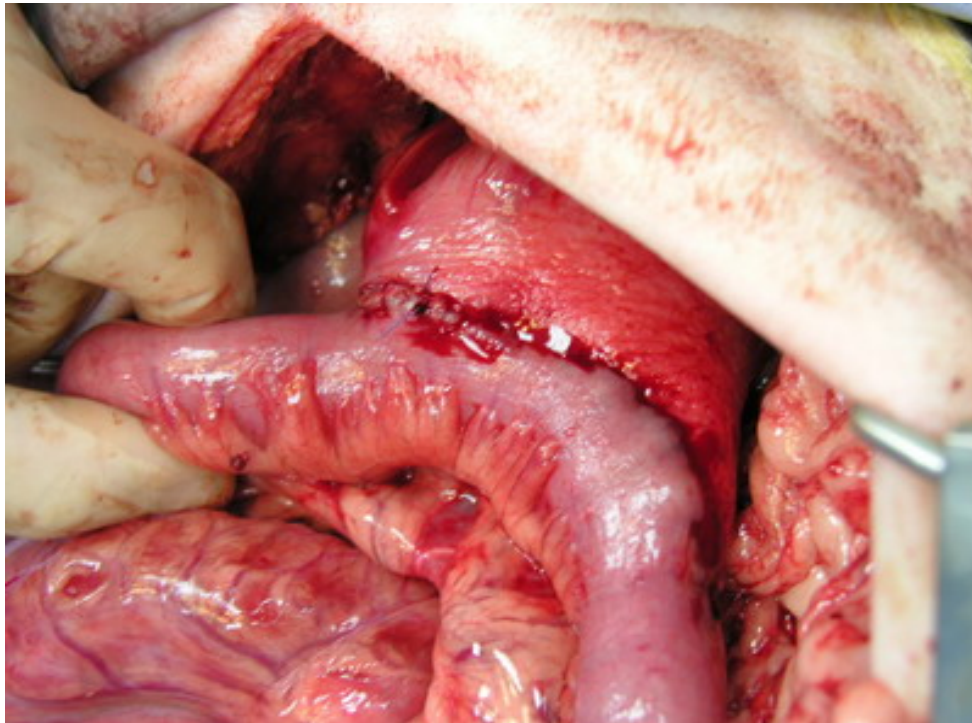
The only potential curative treatment for localised gastric cancer is complete gastric resection. Growth is resectable even if it has involved pancreas, mesocolon or transverse colon but unresectable if portal vein, aorta, base of mesentery, hepatic or celiac artery is involved.

### **Signs of inoperability**

- Malignant ascites
- Jaundice
- Gross cachexia
- Secondaries in rectouterine or rectovesical pouch deposits
- Sister Mary Joseph node
- Krukenberg's tumor
- Virchow's node



## Lymph Nodal Stations



## Billroth 2 gastrectomy

## **Early gastric cancer<sup>17</sup>**

Defined as carcinoma limited to mucosa and submucosa regardless of lymph nodal metastasis. Up to 10 -15% of early gastric cancers are associated with positive lymph node. Endoscopic mucosal resection can be done if tumor is well or moderately differentiated endoscopic type I tumor that is < 2 cm in area or well or moderately differentiated endoscopic tumor, without an ulcer scar that is < 1 cm in area. Conventional gastrectomy with atleast a D1 lymphadenectomy is mandated if submucosal invasion is found after endoscopic mucosal resection.

## **Lymphadenectomy in gastric cancer<sup>47</sup>**

Ideal lymphadenectomy for gastric cancer should accurately stage the extent of disease and predict prognosis; in addition it should have the potential to improve survival by selectively and completely removing all metastatic lymph nodes with minimal morbidity and mortality.

16 nodal stations are grouped according to the location and extension of the primary tumor and the extent of lymphadenectomy is classified according the level of LND (D1-D4) by Japanese Research Society for study of Gastric cancer.

**D1 Dissection:** Perigastric nodes directly attached along the lesser curvature and greater curvatures of the stomach are removed (stations 1-6)

1. Right cardia lymph nodes
2. Left cardia lymph nodes
3. Lymph nodes along the lesser curvature
4. Lymph nodes along the greater curvature
5. Suprapyloric lymph nodes
6. Infrapyloric lymph nodes

**D2 Dissection:** D1 dissection and removal of nodes at stations 7 to 11.

7. Along left Gastric artery
8. Common Hepatic artery
9. Celiac trunk
10. Splenic Hilum
11. Splenic artery

**D3 Dissection:** Includes dissection of lymph nodes at stations 12 through 14, along the hepatoduodenal ligament and the root of the mesentery

12. Lymph nodes in the hepatoduodenal ligament
13. Lymph nodes on the posterior surface of the head of the pancreas
14. Lymph nodes at the root of the mesentery.

**D4 Dissection:** Dissection along para aortic (station 15) and para colic (station 16) region.

## **Advanced Gastric Cancer**

If the growth is not fixed, palliative resection may be done to improve the quality of life and to enhance patient response to chemotherapy. If the palliative resection is not possible, anterior gastrojejunostomy is done to relieve the obstruction.

### **Palliative procedures**

- **Palliative gastrojejunostomy**

Usually done in patients with unresectable antral cancers. Usually anterior gastrojejunostomy is done much away from the tumor.

- **Palliative resection**

Resection is best palliation wherever possible. Usually done in patients with intractable bleeding from tumor area.

- **Devine's exclusion procedure**

If pylorus is fixed and upper part is free from tumor, growth with healthy margins is divided and proximal gastrojejunostomy is done.

### **Adjuvant therapy**

Some form of recurrence develops in most patients who undergo a potentially curative resection for gastric cancers. Although adjuvant therapy is needed in these patients, results have generally been inconsistent.

## **Chemotherapy <sup>1</sup>**

Combination of epirubicin, cis-platinum, and infusional 5 fluorouracil or an analogue such as capecitabine. Same regimen is used as first line chemotherapy for patients with inoperable disease although oxaliplatin is being substituted for cis-platinum.

## **Postoperative external beam radiotherapy <sup>17</sup>**

Radiation therapy is an adjuvant to surgery or combined with sensitising chemotherapy (5-FU). Studies have shown improvement in survival which was attributed to a radiation sensitizing effect of the chemotherapy.

## **Intraoperative radiation therapy <sup>17</sup>**

5 year survival has increased in patients with stage II, III or IV disease. IORT is given to tumor bed for clearance.

## **Neo adjuvant chemotherapy**

Advantages are decreased tumor seeding at surgery, potential opportunity to assess the sensitivity of tumor to chemotherapy, and an improved R0 resection rate. Combination of etoposide, cisplatin, and either 5-FU or doxorubicin is used.



### **Intraperitoneal hyperthermic perfusion**

Adjuvant hyperthermic intraperitoneal chemotherapy of mitomycin C, etoposide and cisplatin resulted in improved survival rates in patients with advanced gastric cancer.

### **Immunotherapy and Hormonal therapy**

Rates of peritoneal recurrence were significantly lower and survival times significantly longer in patients who received chemotherapy and intraperitoneal injection of streptococcal preparation OK-432 than in patients who received chemotherapy alone.

### **Corrosive antral stricture:**

In most cases of corrosive poisoning, esophagus escapes from effect of corrosives and gastric outlet obstruction develops in about 1-6 weeks after ingestion of corrosives. Hydrochloric acid, nitric acid, sulphuric acid, carbolic acid, ferrous sulphate, copper sulphate etc are corrosives causing obstruction.

Surgery is treatment of choice. Antrectomy with gastrojejunostomy can be done. Patients having associated esophageal stricture may be treated with coloplasty.

### **Carcinoma head of Pancreas:**

Two thirds of patients presents with painless progressive jaundice. Usually associated with pruritis, pale coloured stools and dark colored urine. Epigastric pain may radiate to back due to involvement of celiac plexus. Features of malabsorption and steatorrhea may be seen due to exocrine hormone insufficiency. Glucose intolerance may be seen because of altered beta cell function and impaired insulin sensitivity. Symptoms of gastric outlet obstruction may be due to extraneous compression from mass or may be due to direct invasion of pyloric antrum or duodenum.

If the tumor is operable, pancreatico duodenectomy (Whipple's procedure) may be done. If inoperable, palliative triple bye pass (Gastrojejunostomy, Cholecystojejunostomy, Jejunojejunostomy) is done to relieve obstruction.

### **Adult hypertrophic pyloric stenosis:**

Adult hypertrophic pyloric stenosis is characterised by the thickening of the circular pyloric muscle to 2-3 times the normal size with a variable fusiform extension proximally into the antral wall. Longitudinal muscles are not affected by the disease. There may be pyloric thickening noted intraoperatively.

Finney's or Heineke Mikulicz pyloroplasty is done. Ramsted's pyloromyotomy has the risk of producing a diverticulum. Hence not preferred.

### **Advanced carcinoma of Gall Bladder<sup>38</sup>:**

Most common cancer of biliary tree and 6<sup>th</sup> most common cancer of GI tract. More aggressive than cholangiocarcinomas and has poor prognosis. Tumors spread by direct extension into liver segments IV and V and surrounding structures. Duodenal infiltration may present as Gastric outlet obstruction.

Majority of cases are inoperable due to surrounding infiltration. Bypass can be done by Gastrojejunostomy. Patient may be subjected to palliative chemotherapy/ radiotherapy.

### **Gastroduodenal tuberculosis<sup>16</sup>:**

Primary gastroduodenal tuberculosis is rare. The clinical presentation is similar to that of peptic ulcer disease. Usually ulcers occur at junction of 1<sup>st</sup> and 2<sup>nd</sup> part of duodenum. The reported complications include pyloric outlet obstruction, acute ulcer perforation, pyloroduodenal fistula and obstructive jaundice.

Diagnosed by demonstration of giant cells and caseation necrosis in the specimen. Treated by antitubercular drugs if obstruction is partial. Gastrojejunostomy is advised for complete obstruction. Distal partial gastrectomy if multiple TB ulcers are present.

### **Post operative adhesions**

Gastric outlet obstruction can develop due to adhesions following any surgery in liver bed. Commonest being following cholecystectomy because of formation of band between pyloroduodenal junction and liver surface resulting in kinking. Even a surrounding inflammation such as cholecystitis may produce adhesions and cause outlet obstruction.

### **Pseudopancreatic cyst:**

Large cyst may cause compression of pylorus and duodenum resulting in gastric outlet obstruction. Patients may have features of pancreatitis like upper abdominal pain with radiation to back and features of fat malabsorption. Patient may be treated with cystoenterostomy.

### **Bezoars<sup>1</sup>:**

Bezoars are concretions found in the stomach and may result in gastric outlet obstruction.

Trichobezoars (hair balls) are caused by pathological ingestion of hair which remains undigested in stomach. Usually seen in female psychiatric patients often young. Hair ball can lead to ulceration, perforation, gastrointestinal bleeding or obstruction. Treated by removal of bezoar which may require open surgical treatment.

Phytobezoars are made of vegetable matter and found in patients who has gastric stasis often this follows gastric surgery .

### **Foreign bodies of stomach**<sup>28,30</sup>

Most of the ingested foreign bodies causing problems do so in the oesophagus. Once it enters the stomach, uneventful passage through the GIT is seen in 90% of the cases. They sometimes cause gastric outlet obstruction when they get impacted at the pylorus. The patient is usually a young child who presents with abdominal discomfort and vomiting.

The diagnosis is made from the history together with plain X-ray films of the abdomen and can be confirmed by endoscopy. Many of the foreign bodies can be removed by endoscopy. When it fails, laparotomy is done and the foreign body is removed through a gastrostomy incision. If the object is in the duodenum, it is manipulated into the stomach and then removed.

### **Lymphomas of stomach**<sup>1</sup>:

Most prevalent in 6<sup>th</sup> decade of life. Presenting features being pain, weight loss, bleeding and rarely gastric outlet obstruction. Primary lymphomas are B cell derived, tumor arising from Mucosa associated lymphoid tissue. At an early stage disease takes the form of mucosal thickening, which may ulcerate. Diagnosis made by endoscopic biopsy from tumor. Adequate staging is

necessary, primarily to establish whether the lesion is primary gastric lymphoma or part of more generalised process.

Treatment is controversial as some suggest surgery and some as chemotherapy as treatment of choice. Surgery alone is beneficial for patients with localised disease process. Chemotherapy alone is appropriate in patients with systemic disease.

### **Duodenal malignancies<sup>1</sup>**

Although uncommon this is the most common site for adenocarcinoma arising from small bowel. Incidence is maximum in periampullary region and arise in pre existing villous adenomas. Patient may present with obstruction due to polypoid growth of the tumor. Metastasis are common to regional lymph node and to the liver. 70% of patients have resectable disease. Curative treatment involves Whipple's procedure.

### **Wilkie's syndrome<sup>3</sup>**

Also known as Gastromesenteric ileus, Arteriomesenteric ileus, Superior mesenteric artery syndrome. This is an ill defined condition in which 4<sup>th</sup> part of duodenum is compressed between superior mesenteric artery and the vertebral column. Usually occurs in young individuals. Acute presentation is less common and may be precipitated by application of plaster cast or bedrest in supine position. Chronic duodenal ileus presents with epigastric pain, fullness

after meals and foul eructations. Symptoms may get relieved on knee chest position or left lateral position.

Conservative treatment is successful in most cases associated with orthopaedic conditions. In chronic SMA syndrome, if patient fails to respond to conservative treatment then duodenojejunostomy is treatment of choice.

### **Benign tumors of stomach<sup>3</sup>**

Account for less than 2% of all gastric neoplasms. Epithelial polyps are of 2 types, (a)adenomatous which is more common in antrum and (b) hyperplastic, which are distributed throughout the stomach and constitute for 75% of all gastric epithelial polyps. Mesenchymal neoplasms are common in distal stomach and obstruction may be produced by prolapsed of the tumor into duodenum. Leiomyomas are the commonest benign tumor of stomach.

### **Miscellaneous causes of Gastric Outlet Obstruction**

#### **Annular pancreas<sup>1</sup>**

Results due to failure of complete rotation of the ventral pancreatic bud during development. Most often seen in association with congenital duodenal stenosis or atresia and is therefore more prevalent in children with Down's syndrome. Obstruction causes vomiting in the neonate. Treatment is Duodenododenostomy.

### **Pyloric mucosal diaphragm<sup>1</sup>**

Origin of this condition is unknown. It does not become apparent until middle age. Simple excision of the diaphragm is done when pathology is found.

### **Duodenal atresia<sup>1</sup>**

It may be in the form of completely obstructing membrane at the proximal and distal duodenum may be completely separated. Condition may be diagnosed in pre natal ultrasonography of 'double bubble' in fetal abdomen together with maternal polyhydramnios. Most common in children with Down's syndrome. Repair is by duodenoduodenostomy.

### **Complications of gastric outlet obstruction :**

- **Metabolic effects<sup>39</sup>**
  1. Vomiting causes loss of HCl in excess of sodium and potassium.
  2. Hydrogen ions are derived from carbonic acid with residual bicarbonate passing to blood thus causing decreased plasma  $\text{Cl}^-$  and rise in  $\text{HCO}_3^-$ .
  3. Alkalosis is compensated by renal excretion of  $\text{NaHCO}_3$  which maintains the pH level.
  4. Sodium loss caused in long standing cases stimulates aldosterone secretion which conserves sodium at the cost of  $\text{K}^+$  and  $\text{H}^+$



5.  $H^+$  excretion in urine in place of  $Na^+$  in an alkalotic condition is called “Paradoxical aciduria”.

6. Gastric tetany is due to shift of weakly alkaline ionised calcium phosphate to its unionised form in an attempt to reduce alkalosis.

There will be apparent fall in plasma calcium ion levels although total calcium remains normal.

- **Gastritis :** Due to stasis and fermentation of gastric contents
- **Gastric ulcer**
- **Gastric atony:** Excessive peristalsis due to obstruction results in hypertrophy of stomach wall. After this stomach enters a phase of decompensation where it is grossly dilated and atonic.

# INVESTIGATIONS

## **Blood examination**

### a. Haemoglobin

Anemia is seen in majority of the population due to decreased intake and chronic blood loss. In carcinoma stomach there is dimorphic anemia due to presence of both microcytic hypochromic due to loss of blood and megaloblastic due to intrinsic factor deficiency.

### b. Complete haemogram

### c. Blood grouping and typing

Peptic ulcers are more common in patients with 'O' blood group and carcinoma stomach is more common in patients with blood group 'A'.

### d. Liver function tests

To assess liver function in related cases.

### e. Serum electrolytes

There can be hypochloremic, hypokalemic, hyponatremic metabolic alkalosis.

**Urine**

Albumin, sugar and microscopy are done to rule out associated conditions.

**Stool**

To look for occult blood loss, can be black tarry colored in cases of carcinoma stomach.

**Chest X Ray**

To rule out any lung secondaries, for preoperative assessment of lung function and to rule out old Tuberculosis.

**ECG in all leads**

Show changes suggestive of electrolyte imbalance.

**Saline load test <sup>40</sup>**

This was introduced in 1965 by Boyle and Goldstein remains the widely used bed side test for detecting gastric outlet obstruction. Stomach contents are aspirated through a wide bore (18 F) Ryle's tube. 750 ml of normal saline is infused through the tube and is clamped for 30 minutes. Later contents are aspirated, amount of aspirate >400 ml suggests gastric outlet obstruction.

**Upper gastrointestinal endoscopy <sup>1</sup>**

This is done to confirm diagnosis of gastric outlet obstruction and to determine the cause for it. Patients with duodenal ulcer may show cicatrisation of the site of ulcer causing outlet obstruction. In patients with

**CT SCAN SHOWING DIALATED  
STOMACH DUE TO GOO IN CHRONIC DUODENAL ULCER**



carcinoma stomach, helps in taking biopsy from the lesion. The likelihood of a positive yield on biopsy is greater than 95% when 6-10 tissue samples are obtained. It can also be used to retrieve foreign bodies, balloon dilatation and for placement of metallic stents.

### **Ultrasonography**

Helps in determining secondary deposits in liver, ascites, lymph nodal status in cases of carcinoma of stomach. Endoscopic ultrasonography has been used to stage the depth of invasion and regional lymphnode extent in potentially curable cancers.

### **CT Scan <sup>1</sup>**

Presence of stomach wall thickening associated with carcinoma of stomach of any reasonable size can be easily detected by CT scan, but it lacks sensitivity in detecting smaller and curable lesions. Less accurate in detecting T stage of disease as compared to endoluminal USG. Lymph nodes can be detected and depending on size and shape of lymph nodes. Liver metastasis from gastric cancer are less easy to detect using CT

### **Positron Emission Tomography (PET)**

PET is increasingly being used in preoperative staging of gastro-esophageal cancer, as it will often demonstrate occult spread, which renders patient surgically incurable.

### **Contrast radiology (Barium studies)**

Less frequently used as endoscopy is more sensitive investigation for gastric problems. Irregular, persistent filling defects with short history suggests malignancy. Deformed duodenal cap, delayed gastric emptying, dilated stomach are features suggestive of cicatrised duodenal ulcer. Ulcerative growths show an irregular crater with rolled edges of the growth forming a half shadow around the crater (Carman's sign), pancreatic carcinoma may show widened C loop or inverted '3' sign. Adult hypertrophic pyloric stenosis may show bulbous intrusion into the base of duodenal cap. There may be vertical extrinsic compression of third part of duodenum with proximal dilatation in Wilkie's syndrome.

### **ERCP**

Done in patients with obstructive jaundice. There may be sharp duct occlusion in pancreatic cancer.

### **Laparoscopy<sup>17</sup>**

Its particular value is in detection of peritoneal deposits, which is difficult by any other technique unless patient has ascites or bulky intraperitoneal disease. Main limitation is in evaluation of posterior extension of disease.

## **MATERIALS AND METHODS**

The patients for this study has been selected from Govt. Rajaji Hospital, Madurai attached to Madurai Medical College, Madurai during the time period of Sept 2009 to Aug 2011. Overall 80 number of patients has been studied.

Patients presenting with following complaints were included in study.

1. Presence of projectile vomiting of undigested food material, succussion splash heard 3-4 hours after meal, visible gastric peristalsis, presence of mass with above features.
2. Gastric overnight aspirate of >200 ml in fasting state.
3. Positive saline load test: Retention of more than 400 ml of normal saline 30 minutes after administration of 750ml of NS
4. Upper GI scopy (OGD) demonstrating Gastric outlet obstruction.

Detailed history, physical examination and investigation for pre operative assessment was done in all cases. Intra operative findings were noted down and case was followed up in the post operative period.

Gastric contents were aspirated through Ryle's tube after an overnight fast. Saline load test was performed in all cases. 750 ml of Normal Saline infused through Ryle's tube, which was then clamped and was released after half an hour volume of aspirate was noted down. Any volume >400 ml was considered significant.

Upper Gastrointestinal scopy was done in all cases for confirmation of diagnosis. Biopsies were taken wherever required.

Barium meal examination was done in few cases of corrosive oesophageal stricture as the scope couldn't be passed beyond.

Routine investigations like Hb%, Bleeding time, Clotting time, Random blood sugar, Blood urea, Serum creatine, S. electrolytes, blood grouping, urine analysis was done in all cases.

Pre operative dehydration was corrected with intravenous fluids. Gastric decompression was done by continuous drainage of gastric contents through Ryle's tube. Oral fluids were allowed according to the tolerance of patient. Stomach wash was given preoperatively using Normal saline. Anaemia and Hypoproteinemia was corrected using Packed cell and Fresh frozen plasma transfusion.

Anaesthesia: General anaesthesia using Endotracheal tube

Surgery: All findings in intra operative period was noted meticulously.

Post operative period:

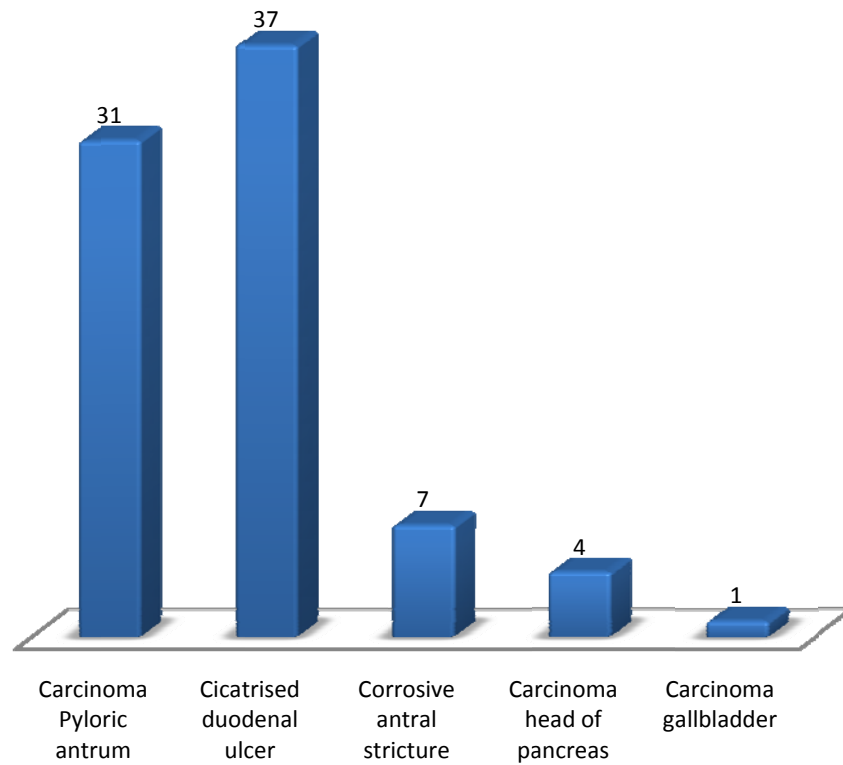
1. Temperature, pulse, blood pressure and respiratory rate chart.
2. Stomach was decompressed using Ryle's tube aspiration.
3. IV fluids were infused until the patients were started on oral fluids.



4. Oral feeds were started after 5<sup>th</sup> post operative day starting with fluids gradually changing to solid foods according to tolerance of patients.
5. Patients were ambulated as early as possible, routine antibiotics were given.
6. All details were recorded in proforma and master chart was made.  
According to the findings in proforma, analysis and discussion carried out and came to an conclusion at the end.

## Causes of gastric outlet obstruction

■ No. of patients



## RESULTS OF OUR STUDY

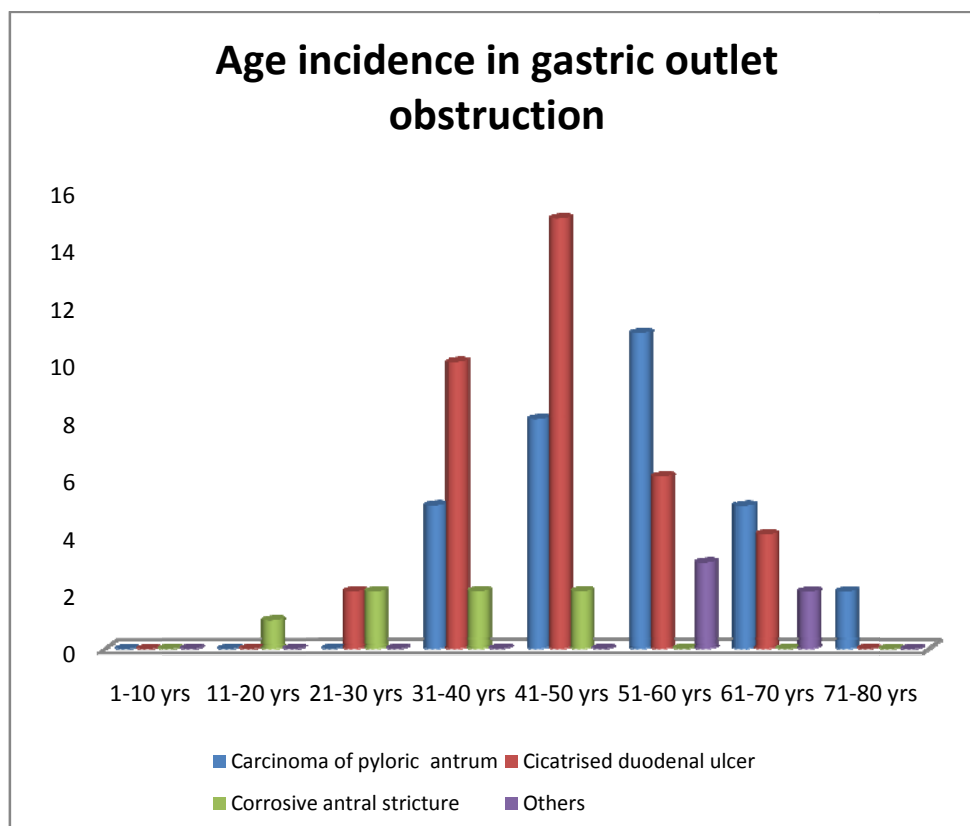
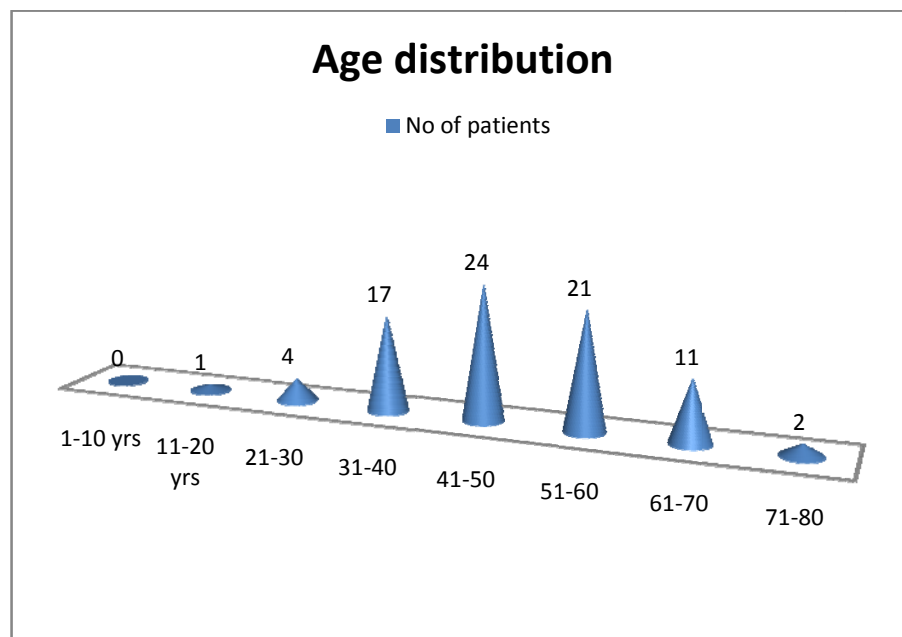
Out of the 80 cases included in our study, 31 patients had carcinoma of pyloric antrum, 37 patients had cicatrised duodenal ulcer, 7 patients had corrosive antral stricture, 5 patients had gastric outlet obstruction due to other causes.

**TABLE No. 1- Causes of Gastric Outlet Obstruction**

<b>Causes</b>	<b>No. of patients</b>	<b>Percentage</b>
Carcinoma Pyloric antrum	31	38.75
Cicatrised duodenal ulcer	37	46.25
Corrosive antral stricture	7	8.75
Carcinoma head of pancreas	4	5
Carcinoma gallbladder	1	2
Total	80	100

### **Age distribution:**

Age incidence in our study ranged from 19 – 75yrs. Incidence of Gastric outlet obstruction was more between 41-50 yrs of age.

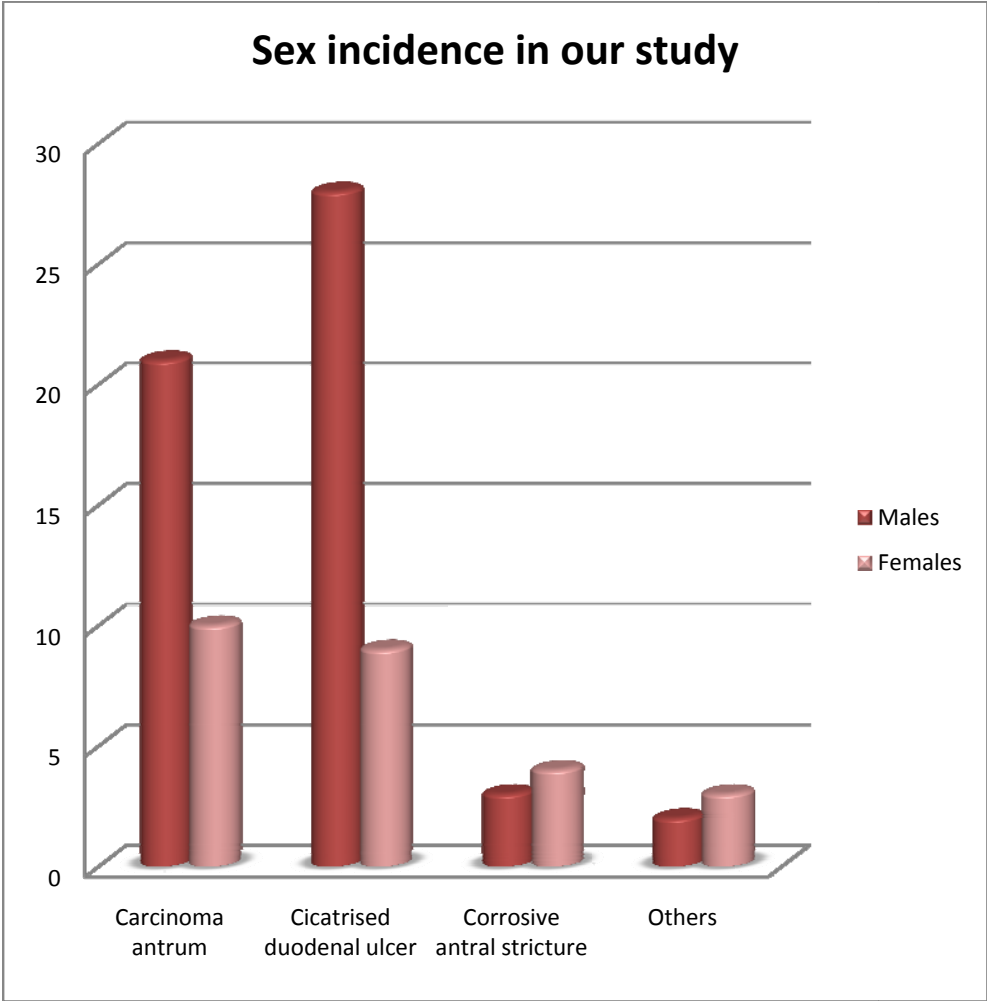


**TABLE No 2: Age distribution**

<b>Age (years)</b>	<b>No of patients</b>	<b>Percentage (%)</b>
0-10	0	0
11-20	1	1.25
21-30	4	5
31-40	17	21.25
41-50	24	30
51-60	21	26.25
61-70	11	13.75
71-80	2	2.5

**TABLE No. 3: Age distribution and causes of Gastric outlet obstruction**

<b>Age group</b>	<b>Carcinoma pyloric antrum</b>	<b>Cicatrised duodenal ulcer</b>	<b>Corrosive antral stricture</b>	<b>Others</b>
1-10	0	0	0	0
11-20	0	0	1 (14.2%)	0
21-30	0	2 (5.40%)	2 (28.5%)	0
31-40	5 (16.12%)	10 (27.02%)	2 (28.5%)	0
41-50	8 (25.80%)	15 (40.50%)	2 (28.5%)	0
51-60	11 (35.48%)	6 (16.21%)	0	3 (60%)
61-70	5 (16.12%)	4 (10.81%)	0	2 (40%)
71-80	2 (6.45%)	0	0	0



Majority of cases of Ca pyloric antrum was noted in the age group 51-60 yrs (35.48%). Obstruction caused due to duodenal ulcer was noted in 41-50 yr age group (40.5%). Youngest case of gastric outlet obstruction due to Carcinoma pyloric antrum was seen in 32 year old male. 19 year old female had gastric outlet obstruction due to corrosive antral stricture.

**Sex distribution:**

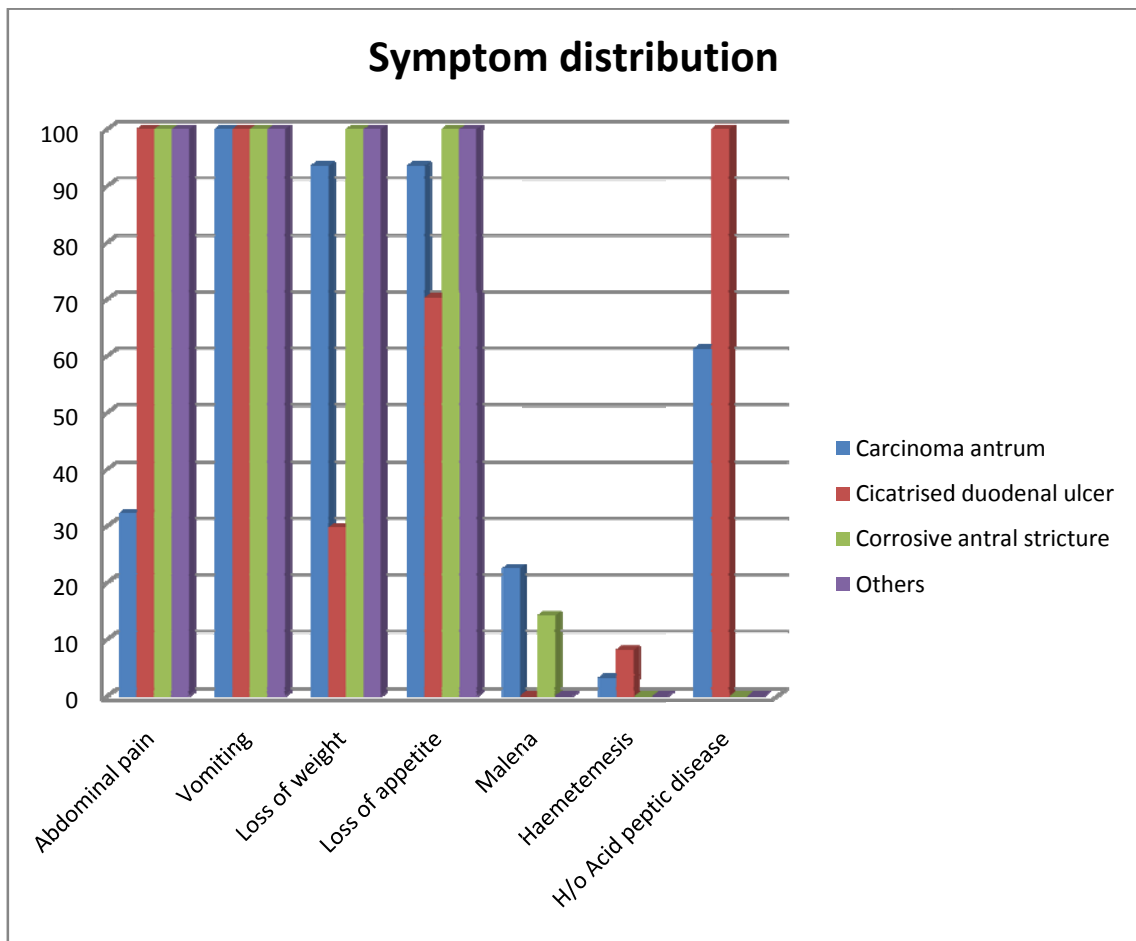
**TABLE No. 4: Sex distribution**

<b>Sex</b>	<b>Total No.</b>	<b>Carcinoma antrum</b>	<b>Cicatrised duodenal ulcer</b>	<b>Corrosive antral stricture</b>	<b>Others</b>
Males	54	21	28	3	2
Females	26	10	9	4	3

Above table shows sex incidence in our study. Majority of patients were males (67.5%). Male to female ratio (M:F) is 2.07:1. M:F in carcinoma antrum was 2.1:1. M:F ratio in cicatrised duodenal ulcer was 3.1:1.

**Socio economic status:** All patients in our study belonged to low socio economic status.

**Smoking:** 62.5% of the patients were smokers all were males. 37.5% were non smokers. Majority of smokers had gastric outlet obstruction due to cicatrised duodenal ulcer.





**Alcohol:** 47.5% of patients were alcoholic and rest were non alcoholic.

**Symptom distribution:**

**TABLE No. 5: Symptom distribution**

<b>Symptoms</b>	<b>Total No.</b>	<b>Carcinoma antrum</b>	<b>Cicatrised duodenal ulcer</b>	<b>Corrosive antral stricture</b>	<b>Others</b>
Abdominal pain	59 (73.75)	10 (32.25)	37(100)	7 (100)	5(100)
Vomiting	80(100)	31(100)	37(100)	7 (100)	5(100)
Loss of weight	52(65)	29 (93.54)	11(29.72)	7 (100)	5(100)
Loss of appetite	66 (82.5)	29 (93.54)	26(70.27)	7 (100)	5(100)
Malena	8(10)	7(22.5)	0	1 (14.2)	0
Haemetemesis	4(5)	1(3.22)	3(8.10)	0	0
H/o Acid peptic disease	56(70)	19(61.29)	37(100)	0	0

Figures in parenthesis show percentage

Above table shows distribution of symptoms among the patients in our study.

Vomiting was predominant symptom in this study. All (100%) patients had vomiting of undigested food material. It was spontaneous and projectile.

Frequency of vomiting was variable and was present occasionally in majority of the cases. The vomitus contained mainly undigested food contents of meals taken earlier.

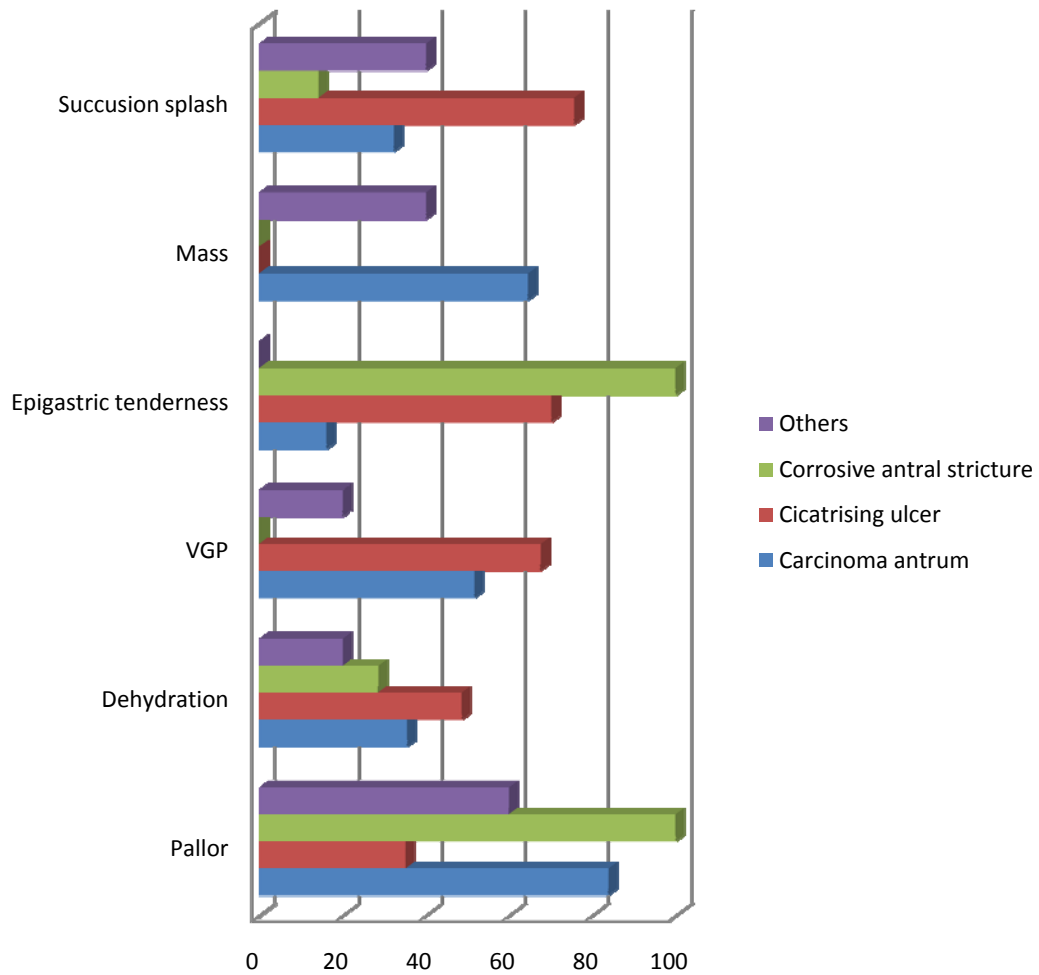
Loss of appetite was the next major symptom in our study group. 93.54% of patients with carcinoma antrum complained of weight loss and anorexia. 70.27% of patients with duodenal ulcer complained of loss of appetite. Weight loss and loss of appetite was present in all cases of corrosive stricture.

Abdominal pain was noted in 73.75% of patients. All 37 patients with duodenal ulcer had pain abdomen, burning in nature, continuous which was relieved by vomiting and was aggravated by food intake. All patients with corrosive stricture had constant pain in upper abdomen which was aggravated by food intake.

History of acid peptic disease was noted in 70% of patients. All 37 patients with cicatrised duodenal ulcer had history of burning sensation of abdomen previously.

Malena was present in 8 patients and haemetemesis in 4 patients.

## Distribution of signs



## SIGNS

**TABLE 6: SIGNS**

<b>Signs</b>	<b>Total No.</b>	<b>Carcinoma antrum</b>	<b>Cicatrising ulcer</b>	<b>Corrosive antral stricture</b>	<b>Others</b>
Pallor	49(69.25)	26(83.87)	13(35.13)	7 (100)	3(60)
Dehydration	32(40)	11(35.48)	18(48.64)	2 (28.5)	1(20)
VGP	42(52.5)	16(51.61)	25(67.56)	0	1(20)
Epigastric tenderness	28(35)	5(16.12)	26(70.27)	7 (100)	0
Mass	22(27.5)	20(64.51)	0	0	2(40)
Succussion splash	41(51.25)	10(32.25)	28(75.67)	1 (14.2)	2(40)

Figures inside parenthesis represent percentage

Anemia was present in 69% of patients. 83.87% of patients with carcinoma stomach had anemia. All corrosive antral stricture patients had anemia.

VGP was seen in 52.5% of patients. Majority (67.56%) of cicatrising duodenal ulcer patients had VGP.

Epigastric mass was palpable in 64.51% of patients with carcinoma stomach.

Succussion splash was noted in 41 patients out of which 28 patients had cicatrised duodenal ulcer.

**Investigations:**

The following investigations were carried out before subjecting the patient for surgery Hemoglobin percentage / Blood routine examination, Urine routine, Random blood sugar, Blood urea, Serum Creatinine, Serum electrolytes, Chest X-ray, ECG, upper G.I. Endoscopy, in all the patients.

Hb % in majority (78.75%) of patients was below 10 Grams.

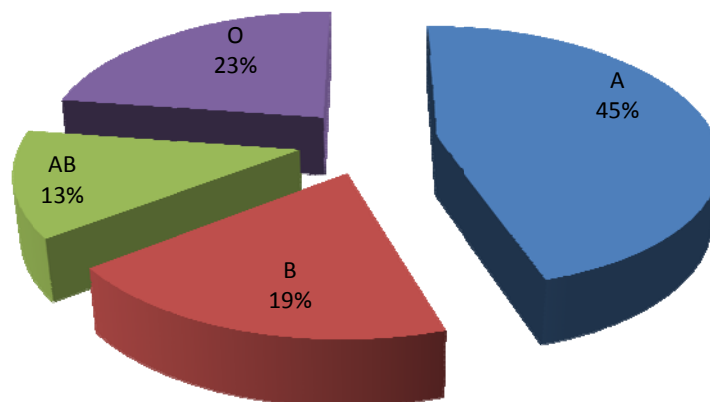
Serum electrolytes were done in all cases. 9 patients had Hypokalemia.

All other blood investigations were normal.

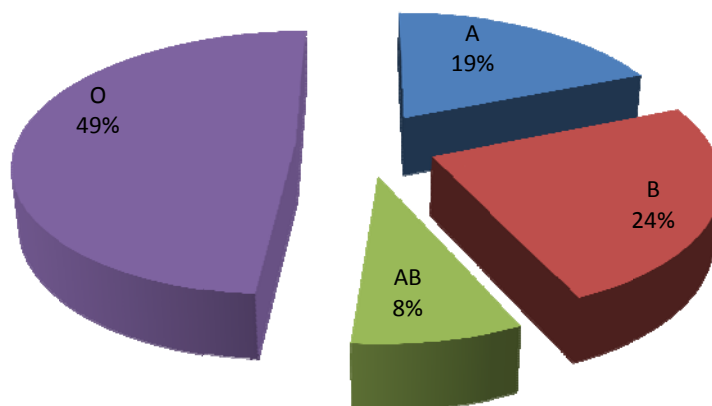
**Distribution of blood group****TABLE 7: Distribution of blood group**

<b>Blood group</b>	<b>Total number of cases</b>	<b>Percentage</b>
A	24	30
B	21	26.25
AB	10	12.5
O	25	31.25
Total	80	100

### Distribution of blood group in patients with carcinoma pylorus



### Distribution of blood group in patients with cicatrised duodenal ulcer



**TABLE 8: Distribution of blood group in patients with carcinoma pylorus**

<b>Blood group</b>	<b>No of cases</b>	<b>Percentage</b>
A	14	45.16
B	6	19.35
AB	4	12.9
O	7	22.58

**TABLE 9: Distribution of blood group in patients with duodenal ulcer**

<b>Blood group</b>	<b>No of cases</b>	<b>Percentage</b>
A	7	18.91
B	9	24.32
AB	3	8.1
O	18	48.6

Majority of patients were having O blood group (31.25%). 30% of patients had A blood group. 48.6% of patients with duodenal ulcer were having O blood group. 45.16% of patients with carcinoma stomach had A blood group.

Upper gastrointestinal scopy was done in all cases mandatorily. All patients with duodenal ulcer sequel showed features of GOO. 12 patients with carcinoma stomach showed Fungating growth in antrum and 19 patients had

prepyloric ulcer/growth. Antral stricture was noted in 5 patients with corrosive acid poisoning. Extraneous compression over duodenum was noted in patients with carcinoma head of pancreas and carcinoma gall bladder. 2 patients with corrosive acid poisoning had esophageal stricture, hence scopy could not be passed beyond.

Barium meal examination was done in 2 patients with corrosive esophageal stricture as upper GI scopy could not be passed beyond stricture.

Ultrasonography was done in all cases of carcinoma pylorus. Liver metastasis was noted in 5 cases. Ultrasound was normal in all duodenal ulcer cases except for two cases which showed gallstones. Carcinoma gallbladder showed ascites in scan.

All patients were evaluated pre operatively and fitness was obtained for surgery. Electrolyte imbalance and anemia was corrected accordingly. Preoperatively patients were kept in liquid diet and supplementary intravenous fluids. Stomach wash was given in the pre operative period using 18 F Ryle's tube with Normal saline.



**TABLE 10: Surgical procedures done**

Procedure	Number of cases	Percentage
<b>Carcinoma antrum</b>		
• Billroth II gastrectomy	9	29.04
• Anterior Gastrojejunostomy	16	51.61
• Anterior Gastrojejunostomy with jejunojejunostomy	1	3.22
• Feeding jejunostomy	4	12.9
<b>Cicatrising duodenal ulcer</b>		
• Truncal vagotomy with posterior gastro jejunostomy	35	94.59
• Truncal vagotomy with posterior gastro jejunostomy with cholecystectomy	2	5.40
<b>Corrosive antral stricture</b>		
• Antrectomy with Billroth II anastamosis	2	28.5
• Antrectomy + coloplasty + feeding jejunostomy	2	28.5
• Anterior Gastrojejunostomy with feeding jejunostomy	1	14.28
• Antrectomy + feeding jejunostomy	1	14.28
• Feeding jejunostomy	1	14.28
<b>Others</b>		
• Triple bypass	4	60
• Anterior gastrojejunostomy	1	20

**Carcinoma pyloric antrum:**

Sixteen patients (51.6%) underwent anterior gastrojejunostomy, 9 patients (29.04%) underwent Billroth II gastrectomy. 4 patients underwent feeding jejunostomy. One patient deferred surgery.

**Cicatrised duodenal ulcer:**

Thirty five patients (94.5%) underwent Truncal Vagotomy with Posterior Gastrojejunostomy as a drainage procedure. Two patients had cicatrised duodenal ulcer with gallstones was treated with truncal vagotomy with gastrojejunostomy and cholecystectomy.

**Corrosive antral stricture:**

Two patients (28.5%) underwent antrectomy with coloplasty and feeding jejunostomy as they had esophageal stricture. 2 patients underwent antrectomy with Billroth II anastomosis.

**Other cases:**

Case no. 35, 36, 46 and 62 had carcinoma head of pancreas underwent Triple bypass as the tumor was inoperable. Patient with Carcinoma gallbladder underwent anterior gastrojejunostomy as a palliative bypass procedure.

In the post – operative period, all the patients were managed with intravenous fluids, antibiotics, Ryle's tube aspiration and analgesics. Oral sips

were allowed after removal of Ryle's tube. Patients were gradually changed over to semi solid and solid diet depending on their tolerance. Sutures were removed after 10<sup>th</sup> postoperative day.

All patients of carcinoma stomach were referred to medical oncology department for further chemotherapy. Follow up was done for a period of 3 months. One patient who underwent coloplasty came with anastamotic stricture which was managed with endoscopic dialatation. Three patients who underwent Truncal Vagotomy and PGJ came with complaints of dumping syndrome, patient was advised diet therapy. Two patients who underwent Billroth II gastrectomy came with complaints of biliary gastritis, which were managed with bile chelating agents.

## DISCUSSION AND ANALYSIS

Discussion is mainly on analysis and observation made regarding presenting symptoms, signs, investigations, operative findings, management and post operative events in 80 cases of gastric outlet obstruction admitted to Govt Rajaji Hospital, Madurai during September 2009 to August 2011.

Out of 50 cases,

Gastric outlet obstruction secondary to carcinoma stomach	--	31
Gastric outlet obstruction due to cicatrised duodenal ulcer	--	37
Gastric outlet obstruction due to corrosive stricture	--	7
Gastric outlet obstruction due to other causes	--	5

**TABLE 11: Etiological factors of Gastric outlet obstruction**

<b>Cause</b>	<b>Our study (%)</b>	<b>Dogo D et al<sup>41</sup>(%)</b>	<b>Harold ellis et al<sup>42</sup> (%)</b>	<b>W H Series<sup>43</sup> (%)</b>	<b>Balint Spence<sup>43</sup> (%)</b>
Carcinoma pylorus	38.75	15	30	36	11.02
Cicatrised duodenal ulcer	46.25	65.7	65	56	80.5
Corrosive antral stricture	14	-	-	-	-
Others	8	18.4	5	8	8.5

Commonest cause of gastric outlet obstruction in our study was cicatrised duodenal ulcer followed by carcinoma of pyloric antrum. All studies conducted previously shows that incidence of gastric outlet obstruction is more due to cicatrised duodenal ulcer. The incidence of obstruction due to carcinoma of pyloric antrum is more common in recent times as per our study results, most probably due to successful treatment of duodenal ulcers by drugs such as proton pump inhibitors.

Most patients affected due to carcinoma of pyloric antrum were in the age group between 5<sup>th</sup> to 7<sup>th</sup> decades. Majority (35.48%) of the patients presenting with this disease were in age group of 51-60 years, youngest age at presentation of this disease was at 32 years.

Maximum incidence of duodenal ulcer sequel patients was noted in age group of 41-50 yrs (40.5%). Youngest age at presentation was at 30 yrs. Male to female ratio noted in our study was 3.1:1 suggesting the disease dominated by males. In series of Fischer et al,<sup>44</sup> men outnumbered women by 2:1.

Our study showed 62.5% of patients were smokers and 47.5% were alcoholics. 75.6% of patients with duodenal ulcer sequel patients were smokers and 56.7% were alcoholics. These values are similar to study results conducted by Donald D Kozoll and Karl A Meyer<sup>37</sup> who reported incidence of alcoholism

and smoking to be 76.2% and 52.3% respectively in their study. This suggests alcohol and tobacco are significant risk factors for causation of duodenal ulcer and carcinoma antrum.

Post prandial vomiting was the main symptom (100%) in all cases of gastric outlet obstruction which was projectile in nature with vomitus being partially digested food material. Loss of appetite (82.5%) and loss of weight (65%) were other major symptoms. Abdominal pain was noted in 73.75% of patients with gastric outlet obstruction. All patients with duodenal ulcer sequel had the symptom of epigastric pain.

**TABLE 12: Incidence of symptoms in cicatrised duodenal ulcer patients**

<b>Symptoms</b>	<b>Present study (%)</b>	<b>Yogiram and Chowdhary <sup>7</sup> (%)</b>	<b>Michael L Schwartz <sup>45</sup> (%)</b>
Abdominal pain	100	87	86
Vomiting	100	80	91
Loss of weight	65	69.2	52
Loss of appetite	82.5	84	-
Constipation	-	23	-

Above table shows percentage of symptoms of duodenal ulcer disease noted in studies conducted by Yogiram and Chowdhary<sup>7</sup> and similar study

conducted by Michael Schwartz <sup>45</sup>. All studies show abdominal pain, vomiting, loss of weight and appetite as the major presenting complaints.

Weight loss was noted in 59.5% of patients in series of Donald D Kazoll and Karl A Meyer<sup>37</sup> and 32% in series of Harvey J Dworken and Harold P Roth<sup>46</sup> suggesting weight loss to be significant in patients with pyloric obstruction.

All patients with carcinoma pyloric antrum had vomiting as their major symptom. Loss of weight (93.54%) and loss of appetite (93.54%) was present in majority of patients. Pain abdomen was not a major presenting feature.

Abdominal pain, vomiting, loss of weight and appetite were seen in all (100%) patients with corrosive antral stricture, carcinoma head of pancreas and carcinoma gallbladder.

All (100%) patients with cicatrised duodenal ulcer had history of acid peptic disease. 61.29% of patients with carcinoma pyloric antrum had history of acid peptic disease. Duration of pain ranges from 2 months to 6 yrs.

Pallor was noted in 61.25% of patients. Majority (83.87%) of patients with carcinoma stomach were anaemic probably due to less amount of nutrition and microscopic blood loss and cancer cachexia.

Visible gastric peristalsis was noted in 67.56% of patients with cicatrised duodenal ulcer. Yogiram and Chowdhary<sup>7</sup> noted the presence of visible gastric

peristalsis in 74% of patients. Visible gastric peristalsis was noted in 51.61% of patients with carcinoma antrum.

In 64.51% of patients with carcinoma antrum epigastric mass was palpable.

Succussion splash was seen in 75.67% of patients with cicatrising duodenal ulcer. Succussion splash was not a major (32.25%) finding in patients with malignancy which is similar to observation made by Harold Ellis<sup>42</sup>.

45.16% of patients with carcinoma pyloric antrum belonged to 'A' blood group. Blood group 'O' was the major (48.6%) group noted in patients with cicatrising duodenal ulcer. This is significant as persons with 'O' blood group are about three times more likely to develop acid peptic disease.

In this study, 51.61% of patients with carcinoma pyloric antrum underwent Anterior gastrojejunostomy as a palliative by pass procedure as tumor was inoperable. 29.04% patients underwent Billroth II gastrectomy. 4 patients underwent feeding jejunostomy.

94.59% of patients with cicatrised duodenal ulcer underwent Truncal Vagotomy with posterior gastrojejunostomy. Two patients had associated gall stone disease, which was treated by Truncal vagotomy with posterior gastrojejunostomy with cholecystectomy.



Two patients with corrosive antral stricture underwent antrectomy with coloplasty and feeding jejunostomy. Two patients underwent antrectomy with Billroth II anastomosis.

Three patients of carcinoma head of pancreas underwent triple bypass procedure. Patient with carcinoma gall bladder underwent anterior gastrojejunostomy.

Ryle's tube was inserted in all patients post operatively for continuous drainage of gastric contents. Oral fluids were started after 5<sup>th</sup> day after removal of ryles tube. Later on patient was changed to solid diet gradually.

All cases of carcinoma stomach, carcinoma head of pancreas and carcinoma gallbladder were referred to Dept of Medical Oncology for further chemotherapy.

One patient who underwent coloplasty came with stricture at the site of anastomosis in neck which was managed by endoscopic dilatation. Three patients who underwent Truncal Vagotomy and PGJ came with complaints of dumping syndrome, patient was advised diet therapy. Two patients who underwent Billroth II gastrectomy came with complaints of biliary gastritis, who were managed with bile chelating agents. Most patients lost follow up.

## **SUMMARY AND CONCLUSION**

The clinical material of the present study includes details of 80 cases of gastric outlet obstruction admitted to Govt. Rajaji Hospital, Madurai during September 2009 to August 2011. A brief introduction and historical review of the incidence of gastric outlet obstruction have been proposed. A detailed review of relevant anatomy, physiology, causes, investigations and management of gastric outlet obstruction has been discussed.

An attempt has been made to compare and discuss the findings in the present series with previously reported literature regarding incidence, clinical symptoms, signs and treatment.

The findings of this study include:

- Most common cause of gastric outlet obstruction is cicatrised duodenal ulcer (46.25%) followed by carcinoma of pyloric antral region (38.75%).
- Incidence of gastric outlet obstruction was more in 5<sup>th</sup> decade in duodenal ulcer sequel, and was more common in 6<sup>th</sup> decade among patients with carcinoma pyloric antrum.
- In 8.75% of patients gastric outlet obstruction was due to corrosive antral stricture following acid ingestion.

- Male to female ratio was 2.07:1 overall. Males to female ratio in duodenal ulcer patients was 3.1:1 and was 2.1:1 in carcinoma pyloric antrum suggesting predominance of disease in males
- Most common presenting complaint was Vomiting (100%), loss of weight and appetite (82.5%) followed by abdominal pain (73.75%). Loss of weight and appetite was more pronounced in patients with malignancy (93.54%) most probably due to chronic loss, decreased intake and because of cancer cachexia.
- Visible gastric peristalsis and succussion splash was noted were less prominent in malignant cases when compared to stenosing duodenal ulcers.
- Majority of the patients with duodenal ulcer and carcinoma stomach were smokers and alcoholics.
- 64.5% of patients with carcinoma had mass palpable per abdomen.
- Majority of the patients with duodenal ulcer sequel were of blood group 'O' and 'A' blood group among patients with carcinoma pyloric antrum.
- All cases of cicatrised duodenal ulcer underwent truncal vagotomy with posterior gastrojejunostomy.
- Majority of patients with carcinoma pyloric antrum tumor was inoperable so 67% of patients underwent palliative procedure. Nine patients underwent definitive surgery.

- Seven patients with gastric outlet obstruction was due to corrosive antral stricture, two patients underwent antrectomy with Billroth II anastomosis and 2 patients underwent antrectomy with coloplasty.
- All 4 patients with carcinoma head of pancreas underwent triple bypass as a palliative procedure as lesion was inoperable. Patient with gallbladder carcinoma underwent anterior gastrijejunosomy as a palliative procedure.

The incidence of gastric outlet obstruction secondary to duodenal ulcer disease has decreased due to

- Availability of highly effective drugs like H2 blockers and proton pump inhibitors.
- Awareness of disease
- Change in food habits.

Truncal vagotomy and gastrojejunosomy is a good procedure for gastric outlet obstruction secondary to cicatrised duodenal ulcer, as recurrence rate and operative mortality is very less. Effective treatment of carcinoma stomach depends on early diagnosis of the disease.

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## PROFORMA

Name: Age/Sex: IP No.:  
Occupation: Income:  
DOA: DOD: Diagnosis: Procedure done:

### CHIEF COMPLAINTS:

- a. Vomiting                      b. Pain abdomen                      c. Anorexia
- d. Mass per Abdomen

### HISTORY OF PRESENTING ILLNESS

- a. Vomiting

Duration                      Frequency:                      Amount:                      Nature:

- b. Abdominal pain

Duration:                      Relation to food:

Aggravating factors:                      Relieving factors:

- c. Mass per abdomen

Duration:                      Site:

- d. Loss of weight and appetite
- e. Malena/Haematemesis
- f. History of Acid peptic disease

### PAST HISTORY

History of any drug intake/any previous illness in past

### FAMILY HISTORY

Any significant history

## **PERSONAL HISTORY**

- a. Dietary habits
- b. Smoking
- c. Alcohol

## **GENERAL EXAMINATION**

- a. Appearance: Built and Nutrition
- b. Pallor:                      Icterus:                      Lymphadenopathy:  
   Dehydration:
- c. Pulse rate:                      Blood Pressure:

## **SYSTEMIC EXAMINATION**

- a. CVS
- b. RS

## **ABDOMINAL EXAMINATION**

- a. Inspection
- b. Palpation
- c. Percussion
- d. Auscultation
- e. PR/PV
- f. Saline load test

## INVESTIGATIONS

### a. Routine Investigations

Hb%:            Blood Sugar:            Blood urea:            Serum Creat:

### Serum Electrolytes:

## Urine examination

## Chest X Ray

ECG

### b. Special Investigations

OGD:

## USG Abdomen

## CT Scan Abdomen

## MANAGEMENT

a. Operative management

Diagnosis:                      Procedure:

Anaesthesia:                      Incision:

### Findings:

HPE:

b. Post operative

Any complications

c. Outcome:

Discharge: Advice:

d. Follow up:

# MASTER CHART

Serial No	Particulars				Symptoms and signs														Investigations						Surgery			
	Name	Age/ sex	IP No.	Diagnosis	Abdominal pain			Vomiting	Loss of weight	Loss of appetite	Malena/hemetemesis	H/o APD	Pallor	Dehydration	VGP	Tenderness	Mass	Succession splash	Smoking	Alcohol	Hb%	OGD	Saline load test	Serum electrolytes	Blood Group	Procedure	Findings	HPE
					present/absent	Aggravating factors	Relieving factors																					
1	Yogammal	50/F	71837	CPA	P	-	-	+	+	+	-	+	+	-	-	-	+	-	-	-	8	FGA	250	N	O	AGJ	Liv Met	ACA
2	Jeyachandran	40/M	44369	CPA	A	-	-	+	+	+	-	+	+	+	-	-	+	-	+	+	8.2	PPG	275	N	O	B II	PPG M	ACA
3	Malaisamy	30/M	90495	CDU	P	+	+	+	-	+	-	+	-	+	+	+	-	+	+	+	9.5	CDU	450	N	AB	TV+PGJ	DS D1	-
4	Dhandapani	37/M	64606	CDU	P	+	+	+	-	+	-	+	-	-	+	+	-	+	+	+	9.2	CDU	350	N	B	TV+PGJ	DS D1	-
5	Chinnu	38/M	71524	CDU	P	+	+	+	+	+	-	+	+	+	+	+	-	+	+	-	8.2	CDU	425	N	O	TV+PGJ	DS D2	-
6	Raman	62/M	88600	CPA	A	-	-	+	+	+	Ma	+	+	-	-	-	+	-	+	+	7.2	PPG	200	N	A	AGJ	PPG F	ACA
7	Muniyandi	57/M	101765	CDU	P	-	-	+	-	-	-	+	-	-	+	-	-	+	+	+	10	CDU	400	N	O	TV+PGJ	DS D1	-
8	Pitchai	50/M	103596	CDU	P	+	+	+	+	+	-	+	+	+	+	-	-	+	+	+	7.9	CDU	350	Ab N	O	TV+PGJ	PPU	-
9	Ganesan	48/M	105487	CDU	P	-	-	+	-	+	-	+	-	-	+	-	-	+	+	-	10.1	CDU	300	N	O	TV+PGJ	PPU	-
10	Rajeshwari	35/F	78632	CPA	A	-	-	+	+	-	-	-	+	+	+	-	-	-	-	-	8.5	PPG	300	N	A	B II	PPG M	ACA
11	Sangaiah	52/M	84809	CDU	P	-	-	+	-	+	-	+	-	-	+	+	-	+	+	+	10.2	PS	410	N	O	TV+PGJ	PS	-
12	Pandi	67/M	87875	CDU	P	-	-	+	-	+	-	+	+	+	+	-	-	-	+	+	8.2	CDU	250	N	A	TV+PGJ	DS D1	-
13	Khadar Moideen	49/M	90972	CDU	P	-	-	+	-	+	-	+	-	-	+	+	-	+	+	+	8.1	PS	420	N	O	TV+PGJ	PS	-
14	Ganesan	39/M	1847	CDU	P	+	+	+	-	+	-	+	-	-	-	+	-	-	+	-	10.2	CDU	250	N	O	TV+PGJ	DS D1	-
15	Bhaskar	45/M	3730	CPA	A	-	-	+	+	+	Ma	+	+	-	-	-	+	-	+	+	8.8	FGA	200	N	A	AGJ	PPG F	ACA
16	Mookammal	34/F	11225	CDU	P	+	+	+	+	+	-	+	+	+	+	+	-	+	-	-	7.2	CDU	450	Ab N	O	TV+PGJ	DS D1	-
17	Pitchai	45/M	14460	CDU	P	+	+	+	+	+	-	+	-	-	+	+	-	+	+	+	10.2	CDU	400	N	B	TV+PGJ	DS D1	-
18	Nagalakshmi	45/F	20668	CPA	A	-	-	+	+	+	-	-	+	-	-	-	+	-	-	-	8	PPG	250	N	A	B II	PPG M	ACA
19	Ramayya	55/M	25296	CPA	P	-	-	+	+	+	-	+	-	-	-	-	-	-	+	+	9.8	FGA	275	N	O	AGJ	Liv Met	ACA
20	Jeyakodi	50/F	30805	CPA	A	-	-	+	+	+	-	-	+	-	+	-	+	-	-	-	7.5	FGA	250	N	A	B II	PPG M	ACA
21	Rathinam	66/M	28527	CPA	A	-	-	+	+	+	-	-	+	-	+	-	+	-	+	+	8.2	PPG	200	N	B	AGJ	Pan Inf	ACA
22	Subbaiah	69/M	36808	CDU	P	+	+	+	-	-	-	+	-	+	+	+	-	+	+	-	11	PS	400	N	O	TV+PGJ	PS	-
23	Arasi	35/F	11583	CDU	P	+	+	+	-	-	-	+	-	-	-	+	-	+	-	-	10.5	DS	420	N	O	TV+PGJ	DS D1	-
24	Ganesan	41/M	10315	CDU	P	-	-	+	-	-	-	+	-	-	+	-	-	-	+	-	10.2	CDU	300	N	B	TV+PGJ	DS	-
25	Chinnan	45/M	42231	CDU	P	-	-	+	-	+	-	+	+	-	+	-	-	+	+	+	8.6	CDU	350	N	O	TV+PGJ	CDU	-
26	Chellamal	60/F	47827	CPA	A	-	-	+	+	+	-	-	+	-	-	-	+	-	-	-	8.2	FGA	250	N	A	FJ	Pan Inf	ACA
27	Karuppusamy	45/M	46144	CAS	P	+	+	+	+	+	Ma	-	+	+	-	+	-	+	+	+	8.3	Ant Str	200	N	B	FJ	Ant Str	-
28	Matchakalai	32/M	46638	CPA	A	-	-	+	+	+	-	+	-	-	-	-	+	-	-	-	10.5	PPG	200	N	AB	FJ	Liv Met	-
29	Ayyamal	65/F	60445	CPA	A	-	-	+	+	+	-	+	+	+	+	-	+	-	-	-	8.6	FGA	250	Ab N	A	AGJ	Pan Inf	-
30	Kalyani	40/F	61749	CPA	A	-	-	+	+	+	-	-	+	-	+	-	-	+	-	-	8.9	FGA	400	N	B	AGJ	LP++	ACA

31	Chinnakurupayya	45/M	50869	CAS	P	+	+	+	+	+	-	-	+	-	-	+	-	-	+	+	7.1	Ant Str	225	N	B	AGJ+FJ	Ant Str	-
32	Nallayya	63/M	52340	CPA	A	-	-	+	+	+	-	+	+	-	+	-	-	-	+	+	7.2	PPG	300	N	A	B II	PPG M	ACA
33	Subbayya	51/M	52363	CPA	P	-	-	+	+	+	-	+	+	-	-	-	-	+	+	-	8.2	PPG	350	N	AB	AGJ	Liv Met	ACA
34	Rajendran	51/M	54682	CPA	A	-	-	+	-	+	-	+	+	-	-	-	+	+	+	-	8.2	FGA	425	N	O	Pt defe	-	ACA
35	Mallika	51/F	69707	CHP	P	-	-	+	+	+	-	-	-	-	-	-	+	-	-	-	11.2	Extr Com	250	N	A	TrBp	Vas+	ACA
36	Rajathi	70/F	65193	CHP	P	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	10.5	Extr Com	250	N	B	TrBp	Liv Met	ACA
37	Periyasamy	60/M	56612	Ad CS	A	-	-	+	+	+	Ma	+	+	+	-	-	+	+	+	+	8	FGA	300	N	A	AGJ+JJ	Pan Inf	ACA
38	Rajagopal	35/M	60430	CAS	P	+	-	+	+	+	-	-	+	+	-	+	-	-	+	+	8.6	Ant Str	250	N	B	Antr+FJ	Ant Str	-
39	Govindaraj	63/M	60094	CPA	A	-	-	+	-	-	-	+	+	-	+	-	-	-	+	-	8.5	PPG	300	N	A	AGJ	Liv Met	ACA
40	Jameela Biwi	50/F	33384	Ad CS	A	-	-	+	+	+	-	+	+	-	-	+	-	-	-	-	8.2	FGA	300	N	A	AGJ	Pan Inf	ACA
41	Malli	42/F	14200	CDU+GS	P	+	+	+	-	-	+	-	+	+	+	-	+	-	-	-	10.3	PS	450	N	O	TVGJ+ C	PS+GS	-
42	Marimuthu	55/M	59451	CGB+GOO	P	-	-	+	+	+	-	-	+	-	+	-	+	+	+	+	8.7	Extr Com	320	N	AB	AGJ	Vas+As	PACA
43	Vimala	30/F	90301	CAS+OS	P	+	+	+	+	+	-	-	+	-	-	+	-	-	-	-	6.9	OS	-	N	AB	Antr+FJ+Co	Ant Str	-
44	Ponnukala	35/F	87952	CAS	P	+	+	+	+	+	-	-	+	-	+	+	-	-	-	-	7.2	Ant Str	250	N	A	Antr+BII	Ant Str	-
45	Subbayya	53/M	65301	Ad CS	A	-	-	+	+	+	-	+	+	+	-	+	-	+	+	+	7.9	FGA	300	Ab N	O	FJ	Post Inf	ACA
46	Chellammal	60/F	54373	CHP	P	-	-	+	+	+	-	-	+	+	+	-	-	+	-	-	8.6	Extr Com	300	N	A	TrBp	Vas+	ACA
47	Umamaheshwari	24/F	70074	CAS	P	+	+	+	+	+	-	-	+	-	-	+	-	-	-	-	8	Ant Str	250	N	B	Antr+BII	Ant Str	-
48	Dharani	75/F	81365	CPA	A	-	-	+	+	+	-	+	+	+	-	+	-	-	-	-	8.5	PPG	375	N	B	FJ	LP++	ACA
49	Lakshmi	19/F	88880	CAS+OS	P	+	+	+	+	+	-	-	+	-	-	+	-	-	-	-	8	OS	-	N	AB	Antr+Colo	Ant Str+OS	ACA
50	Kandasamy	55/M	24738	CPA	A	-	-	+	+	+	-	+	+	-	-	-	-	-	+	-	7.2	FGA	200	N	A	AGJ	Liv met	ACA
51	Venkatasamy	48/M	34036	CPA	P	-	-	+	+	+	MaH	+	+	-	+	+	+	+	+	+	7	PPG	400	N	O	B II	PPG M	ACA
52	Chellam	55/M	36038	CDU	P	+	+	+	+	+	H	+	+	+	+	+	-	+	+	+	6.5	CDU	420	N	O	TV+PGJ	PS	-
53	Mottaiyan	68/M	52824	CDU	P	+	+	+	+	+	-	+	-	-	-	+	-	-	+	+	10.2	CDU	250	N	AB	TV+PGJ	DS D1	-
54	Nagavalli	40/F	52802	CDU	P	+	+	+	-	+	-	+	+	+	+	+	-	+	-	-	8	PS	430	Ab N	A	TV+PGJ	PS	-
55	Sasikumar	30/M	73035	CDU	P	+	+	+	-	-	-	+	-	-	-	+	-	-	+	-	10.1	PS	250	N	A	TV+PGJ	PS	-
56	Lalitha	58/F	71093	CDU	P	+	+	+	-	-	-	+	-	+	+	+	-	+	-	-	10.6	CDU	300	N	B	TV+PGJ	DS D1	-
57	Lakshmi	65/F	23132	CDU+GS	P	+	-	+	+	+	-	+	+	-	-	-	-	+	-	-	8.1	CDU	420	N	A	TVGJ+ C	CDU	-
58	Vishwanathan	50/M	29881	CDU	P	+	+	+	-	+	-	+	+	+	+	+	-	-	+	+	8.5	PS	250	N	A	TV+PGJ	PS	-
59	Chinnammal	58/F	34901	CPA	A	-	-	+	+	+	-	-	+	+	-	+	+	+	-	-	8.8	PPG	350	Ab N	B	B II	PPG M	-
60	Neelamegham	45/M	34704	CDU	P	+	+	+	-	+	-	+	-	+	+	+	-	+	+	+	10	PS	380	N	B	TV+PGJ	PS	-
61	Muthaiah	44/M	73123	CDU	P	+	+	+	+	-	H	+	+	-	-	-	-	+	+	+	7	DS D2	420	N	A	TV+PGJ	DS D2	-
62	Karupayya	65/M	75896	CHP	P	-	-	+	+	+	-	-	+	-	-	-	-	-	-	-	8	Extr Com	280	N	B	TrBp	Vas+	ACA
63	Senthil Murugan	37/M	84612	CPA	P	-	-	+	+	+	-	-	-	-	+	+	-	-	+	+	10	PPG	250	N	O	AGJ	Pan Inf	ACA
64	Alagumalai	55/M	84623	CDU	P	+	+	+	-	+	-	+	-	-	+	+	-	+	+	+	9.8	PS	400	N	B	TV+PGJ	PS	-
65	Bose	55/M	98152	CDU	P	+	+	+	+	+	-	+	-	+	+	+	-	+	-	-	10.4	PS	410	Ab N	O	TV+PGJ	PS	-
66	Ramasamy	41/M	89527	CPA	A	-	-	+	+	+	Ma	+	+	+	-	-	+	+	+	+	6	PPG	300	N	B	AGJ	PPG F+Pan Inf	ACA
67	Lakshmi	42/F	10167	CDU	P	+	+	+	-	-	-	+	-	+	+	-	-	-	-	-	9.5	PS	290	N	O	TV+PGJ	PS	-
68	Nagaraj	40/M	102657	CDU	P	+	+	+	-	+	-	+	-	-	+	-	+	+	+	+	9.6	PS	450	N	AB	TV+PGJ	PS	-
69	Mariammal	50/F	44152	CDU	P	+	+	+	+	+	H	+	+	+	-	+	-	-	-	-	6.8	PS	200	N	B	TV+PGJ	PS	-
70	Mayandi	50/M	50206	CDU	P	+	+	+	-	-	-	+	+	+	+	-	-	+	+	+	6	DS D1	360	Ab N	O	TV+PGJ	DS D1	-
71	Mohammed Ali	73/M	50204	CPA	P	-	-	+	+	+	-	-	+	-	-	+	-	+	+	+	10.2	PPG	290	N	B	B II	PPG M	ACA
72	Rajaram	55/M	1579	CPA	P	+	+	+	+	+	Ma	+	+	+	+	+	+	+	+	+	6.5	PPG	350	N	AB	AGJ	PPG+As	ACA
73	Mookayya	47/M	9653	CPA	P	+	+	+	+	+	-	-	-	-	-	-	-	+	+	+	9.5	PPG	270	N	A	AGJ	PPG F	ACA
74	Alagar Pandi	33/M	15019	CDU	P	+	-	+	-	-	-	+	-	-	+	+	-	+	+	+	9.6	PS	300	N	B	TV+PGJ	PS	-
75	Muthappa	60/M	13243	CPA	P	-	-	+	+	+	-	-	+	+	-	-	-	-	+	-	8	PPG	270	N	AB	AGJ	PPG+As	ACA

76	Rakammal	40/F	46492	CDU	P	+	+	+	-	+	-	+	-	+	+	+	-	+	-	-	9.7	DS D2	400	Ab N	B	TV+PGJ	DS D2	-
77	Chellam	40/M	48089	CDU	P	+	+	+	-	-	-	+	+	-	-	+	-	+	+	+	8.4	DS D1	420	N	O	TV+PGJ	DS D1	-
78	Pandi	59/M	48683	CPA	P	-	-	+	+	+	Ma	+	+	+	+	+	+	-	-	-	7	PPG	250	N	A	B II	PPG M	ACA
79	Raja	45/M	17463	CDU	P	-	-	+	-	+	-	+	-	-	-	-	-	-	-	+	9.8	PS	225	N	A	TV+PGJ	PS	-
80	Pitchai Mani	47/M	48744	CDU	P	+	+	+	+	+	-	+	-	+	-	+	-	+	+	+	9.6	DS D1	300	N	O	TV+PGJ	DS D1	-





## KEY TO MASTER CHART

M	–	Male
F	–	Female
+	-	Present
-	-	Absent
P	–	Present
A	–	Absent
Ma	-	Malena
H	-	Haematemesis
CPA	-	Carcinoma pyloric antrum
CDU	-	Cicatrised duodenal ulcer
CAS	-	Corrosive antral stricture
CHP	-	Carcinoma head of pancreas
Ad CS-		Advanced carcinoma stomach
CDU+GS-		Cicatrised duodenal ulcer+Gall Stones
CAS+OS-		Corrosive antral stricture+oesophageal stricture
CGB+GOO-		Carcinoma gall bladder+gastric outlet obstruction
FGA-		Fungating growth antrum
PPG-		Pre pyloric growth
PPU-		Pre pyloric Ulcer
PS -		Pyloric Stenosis
DS -		Duodenal Stenosis
DS D1-		Duodenal Stenosis at 1 <sup>st</sup> part

DS D2-	Duodenal Stenosis at 2 <sup>nd</sup> part
Ant Str-	Antral Stricture
Extr Comp-	Extraneous Compression
OS -	Oesophageal Stricture
N -	Normal
Ab N-	Abnormal
AGJ-	Anterior Gastro jejunostomy
B II-	Billroth II gastrectomy
TV+PGJ-	Truncal Vagotomy +Posterior Gastro jejunostomy
FJ -	Feeding Jejunostomy
TrBp-	Triple Bypass
AGJ+JJ-	Anterior Gastro jejunostomy+ Jejunojejunostomy
Antr+FJ-	Antrectomy+Feeding Jejunostomy
Colo -	Coloplasty
Liv Met-	Liver Metastasis
PPG M-	Pre pyloric growth mobile
PPG F-	Pre pyloric growth fixed
Pan Inf-	Pancreas Infiltration
Ant Str-	Antral Stricture
LP++ -	Liver and pancreas infiltration
Vas+ -	Vascular involvement
PV inv+A-	Portal vein invasion+ ascites
Post inf-	Posterior Infiltration
ACA -	Adenocarcinoma